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## ORIGINAL ARTICLES.

### ADDRESS ON PHYSIOLOGY.

Delivered in the Section of Physiology and Dietetics, at the Forty-third annual meeting of the American Medical Association, held in Detroit, Mich., June, 1892.

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*Gentlemen:* It is obviously the first and at the same time the most pleasant duty of your chairman, to congratulate the members of this Section on the fact, that the American Medical Association, at its forty-third annual meeting decided by an almost unanimous vote, to "omit the word physiology from the title of the Section on Practice of Medicine, and to form a new Section on Physiology and Dietetics." The wisdom of this action admits of no doubt, for the creation of this new Section enables those more especially interested in our branch of the medical sciences to present and discuss the results of their labors in this field under conditions much more favorable and satisfactory than could possibly be afforded while physiology was coördinated in the same Section with a branch, in which the usually superabundant, important and practical matters presented for consideration were apt to encroach upon those of a more specifically physiological bearing.

The next duty to be performed is to express to you the high appreciation of the honor conferred by being selected the first chairman of this important Section: yet, this appreciation is weighed with the feeling of regret, that the distinction was bestowed upon one, who but too well knows that he lacks many of the qualifications for his position. He hopes, however, that with the kind coöperation of his fellow members, his task will be rendered less weighty, and has the assurance that the results of your work will bear out the wisdom of the Association in the creation of the new Section.

Now, according to a by-law of our Association, it is made the function of the Chairman, to review in an address to be presented on the first day of the annual meeting, the recent advances made in the branches belonging to his Section, and in compliance with this rule, I now have the honor to present to your consideration a brief resumé of topics directly concerning us as physiologists. But in looking over the field of physiology as a whole, we find so much to claim a more than passing attention; so much active cultivation in every direction by so great a number of diligent investigators, that the task of presenting even a very concise synopsis of the work done in every part of the field appears altogether beyond the power of your reporter, in view of the vast amount of material accumulated in the literature of the year, an abundance so great as to become a very *embarras de richesses*. Hence it has been deemed advisable to confine the selection to the progress made in the physiology of the nervous system, partly because of a personal predeliction, but mainly because of the full recognition of the fact, that the activity of the nervous system underlies, regulates, and modifies directly or indirectly all vital functions whatsoever. Thus any discussion or investigation of the functions of the *working organs* so-called, can never be complete, unless allied to a consideration of the force calling them into play, to an investigation of the nerve-mechanism exciting, inhibiting or in some other way modifying them; and it has been said with perfect truth, that a complete history of the physiology of the nervous system would almost represent a history of physiology in general. In thus selecting, what appears to him the most interesting branch of physiology, your reporter can not, at the same time, close his eyes to the fact, that he is about to engage in the recital of what must necessarily prove a twice told tale to his hearers, and he also feels, that he runs the great risk of omitting to mention topics, that may possess the highest value in the opinions of some, while laying what may be called undue stress on those, that in the eyes of others may seem to be entitled to less consideration. Trusting, however, to your kind indulgence, he hopes that the imperfect sketch about to be offered may contain at least some food for thought, and a few points of interest to the members of the Section.

The functions of the cerebro-spinal axis as a whole are discussed in his usual brilliant style by Richardson (*Asclepiad*, 1891, *p.* 365 *et sequ*), in a lecture before the Medico-Psychological Association, in which he argues in support of the theory, that the central nervous system is to be regarded in the light of a static thermal centre and water-power; the mainspring of all vital actions. That the gray matter of the cerebro-spinal axis is the seat of a slow combustion, while the white matter supplies the combustion material, and that this accounts for the grand nerve phenomena of life in action, and their repose in sleep, both states depending on variations of tension.

It is also claimed for the theory, that it explains the effects of varying external pressures and temperatures on the central nerve-organism. The author assigns to the cerebro-spinal fluid the most important function, holding it to be the condensed fluid of the combustion of the cerebro-spinal axis, which latter is not merely an absorbing centre for the reception of external vibrations, but a true chemical and dialysing centre and the centre of static combustion. The theory looks upon the ganglia as supplementary centres supplied from the main source, while plexuses are inter-communicating points of nerves to enable vibrations to be carried on, should one or more nerves of a plexus fail to functionate, while decussating fibres are the means by which centres are prevented from becoming independent of each other and

losing their compensating balance. There are two distinct combustions in the body. The one static, in the central nervous system, the other in the muscular and active vital organs, yielding the animal heat recognized as the sensible heat of the body. The latter, while apparently independent and more active, may possibly depend for its continuous existence on the slower cerebral combustion, that keeps it alight and regulates its activity by regulating its blood supply, and through this its oxygen and combustible material.

This theory has at least the charm of being well presented, with the additional merit of attempting an explanation of nerve-function upon a purely physical and chemical, *i.e.*, scientific basis, and while indeed, like all other theories upon nerve force, it fails in unveiling this great mystery, the method adopted by its author is the only one that promises a rational solution of the question, for it is the only method to be applied in modern physiology, and to which our science owes every advance that has been made in an elucidation of the problem of life.

Beard (*Northwestern Lancet*, 1891, pp. 181, *et sequ*), after a resumè of the theories of sleep as advanced by Preyer and others, reaches the conclusion that sleep appears to be the result of a fall of temperature incident to the decline of destructive metabolism in the nerve centres, while insomnia is the result of a maintenance of a pseudo-active metabolism and of consequent temperature in these cases. The practical conclusions to be drawn from this theory are, that those remedial measures which directly diminish metabolism and thus reduce cerebral temperature best afford immediate relief from insomnia; that those agents indirectly producing this result through their influence on the vascular system, are of secondary value only, while those permanently improving nutrition, stimulating waste-removal and restoring the normal ebb and flow of metabolic activity in the nerve cells: electricity, massage, etc., have the largest field of usefulness in the cure of insomnia.

The sense of muscular effort is treated at length by Waller (*Brain*, 1891, p. 179), who sums up his views by holding that the duly coördinate action of the muscular execution depends upon three factors: coördination at the central origin of action, resulting from past experience and consisting in the emission of nerve impulses grouped and measured in conformity with intended and foreseen effects. Appropriate reflex response, of which the immediate causes are centripetal impulses from sense organs, including those from the skin, muscles, and articulations, collectively referred to as muscular sense. Direct muscular response to passive stretching and extensive vibrations.

Belmondo and Oddi, (*Arch. Ital. de Biol.*, 1891, p. 17) offer experimental researches of great interest, on the influence of the posterior spinal roots upon the excitability of the anterior roots with the following important results: 1. In destroying the conductivity of the posterior roots by means that cause no irritation whatever, there is found a constant diminution in the excitability of the corresponding anterior roots. 2. Every excitation of the sensory roots per contra, increases during a longer or shorter period, the excitability of the anterior roots. Sometimes this hyper-excitability is preceded by a period, generally very brief, during which the excitability of the anterior roots is lost or greatly diminished. 3. These different factors seem independent of cerebral influence and consequently of consciousness, at least in the proper sense of that term. This influence is not affected by section of the medulla oblongata. The authors hold, that stimuli are constantly reaching the central nervous system from the periphery and pass into the motor roots.

In this connection may be noted the investigations of Schaffer (*Arch. f. Mikrosk. Anat.*, 1891, p. 157) upon the arrangement of fibres in the spinal cord. undertaken in blind worm, grass-snake, tortoise, lizard, rabbit, bat and cat, proving that fibres from both sides of the posterior horn go directly through the anterior commissure into the opposite anterior column. Fibres also pass from the lateral columns into the opposite anterior column.

Our knowledge of the physiology of the cranial nerves has received some important additions; thus Bernheimer, in a very valuable study of the origin of the optic nerve, made upon the brain of the human fœtus, and of the new born, shows that the external corpus geniculatum is not merely a ganglion *through* which the fibres of the optic tract pass, but that it is a true place of origin for many of these fibres. That the second bundle of tractus-roots, perhaps the largest bundle of all, comes from the corpus subthalamicum. That a few fibres are derived from the internal geniculate ganglion, while two fibre-tracts can be demonstrated as coming from the thalamus opticus, viz., a deep and a superficial root. Now the importance of these researches lies in this, that they show that we have hitherto had an exaggerated idea of the significance of the corpora quadrigemina as points of origin of the optic nerve, for while the author thinks that there is a root derived from the posterior corpora quadrigemina, he appears to doubt the existence of a root derived from the anterior corpora quadrigemina. He also failed to prove the existence of the so-called descending root; of the origin from the oculo-motor nucleus and from the crus cerebelli. These results, if found correct, must modify our views upon the functions of the subthalamic bodies, optic thalamus and corpora quadrigemina as relating to vision.

Hebold (*Neurol. Centralbl.*, 1891, p. 167) concludes from pathological data, that the fibres in the optic nerve pass in closed bundles; the uncrossed bundle lies on the outer side of the optic nerve, chiasm and tract, while the crossed bundle is central in the nerve, but ventral in the tract.

The *oculo-motor* nerve is generally looked upon as the only nerve of accommodation, but Morat and Doyon (*Arch. de Physiol. norm. et pathol.*, 1891, p. 507) offer experimental proof in favor of the view, that instead of one, there are in reality two nerves of accommodation: the oculo-motor for near vision, and the sympathetic for distant vision; and that the latter acts as an inhibitory nerve to the former, hence the cervical sympathetic, carrying these fibres to the eye is the inhibitory nerve of accommodation.

In connection with the trigeminus we find an interesting point in an experiment by Gley (*Compt. rend. Soc. de Biol.*, 1891, p. 173). He presented before the Société de Biologie a rabbit, in which intracranial section of the ophthalmic division of the trigeminus, together with that of the oculo-motor, was not followed by trophic changes in the eye, although that organ was completely anæsthetic. This is ad-

ditional proof against the theory that trophic lesions following sections of the V. are due to irritation of foreign bodies and loss of sensibility in the parts supplied by the nerve.

From a study of a case of bulbar paralysis and the areas of degeneration in the medulla as shown by the microscope, Tooth and Turner (*Brain*, 1891, p. 473) present some interesting and important conclusions on the cerebral origin of certain cranial nerves. These investigations support the views of Mendel, derived from experiments on rabbits, and guinea-pigs, that the oculo-facial group of muscles receives its nerve supply from the oculo-motor nucleus by way of the facial. They also show that the oro-facial group (orbicularis oris) is presumably innervated by the hypoglossal nucleus; that the motor fibres for the palate and larynx carried in the accessorius vagi are in all probability derived from the region of the hypoglossus nucleus. These results are not only of interest in point of diagnosis and pathology of bulbar paralysis, but equally interesting from a physiological point of view, as illustrating the rule, that muscles usually or habitually acting in harmony receive their motor supply from the same central source, although these fibres may in part be carried to them by different peripheral nerve tracks.

The old controversy as to the seat of origin of the auditory nerve has led Sala[1] to new experimental researches with a view of settling this mooted point, and he feels himself justified in the conclusion, that neither Deiter's nor the dorsal, nor Bechterew's nucleus have any claim to be considered points of origin of the auditory nerve, but that the anterior nucleus and the lateral tubercle are the real and only nuclei of origin, the former for the anterior, the latter for the posterior division of the nerve.

Howel and Huber[2] state that the communicating branch between the superior and inferior laryngeal nerves (Galen's anastomosis) does not carry depressor fibres from the heart, but that, as proven by experiment and secondary degeneration in the dog, it is a sensory branch of the superior laryngeal for the trachea and œsophagus, although fused with the inferior laryngeal in part of its course. This proves Longet's supposition to have been correct, who believed that the œsophagus received a sensory nerve supply from the laryngeal through this anastomosis.

Our knowledge of the function of the phrenic nerve has been increased by Ferguson (*Brain*, 1891, p. 282), who adduces conclusive proof, experimental as well as pathological, that the phrenic nerve is not, as generally held, a purely motor, but a mixed nerve, thus supporting the opinion expressed by Ross from a clinical standpoint, and the suggestion made by Peter and Henle.

The *sympathetic* system has received considerable attention at the hands of physiological investigators. Thus Arloing (*Arch. de Physiol.*, 1891, p. 160) records the continuation of his researches on the functions of the cervical sympathetic, which lead him to the conclusion that the cervical sympathetic contains trophic fibres. The experiments were made on the ox and dog. The muzzle of the former showed marked histological changes in the papillæ and stratum granulosum after section of the nerve. In the dog the changes were not immediate, because of absence of glands in the more hairy parts of its nose, which are abundant in the ox. But the changes were marked after two months, the skin of the nose becoming papillated and dry, with great hypertrophy of the corneous layer. He interprets these results by concluding that the cervical sympathetic supplies trophic fibres to the glands and epithelium, and that their action is independent of that of the vaso-motor and glandular fibres.

The same author (*Journ. de Méd. veter. et Zooteich*, 1891, p. 171), contributes researches upon the secretory functions of the cervical sympathetic as affecting the activity of the lachrymal, sebaceous and sudoriparous glands, and finds that its action is inhibitory to lachrymal secretion, but excitor to the sebaceous and sudoriparous glands.

Sherrington (*Brit. Med. Journ.*, 1891, p. 635) finds that the excitation of the cervical sympathetic in the monkey by feeble induction shocks thrown into the peripheral end of the divided nerve of one side (the animal being under an anæsthetic), results in a slight enlargement of the palpebral aperture by a lifting of the upper lid, dilatation of the pupil, a drawing back of the pinna of the ear, with slow erection of the skin of the nostril and hair of forehead, temple, cheek and upper part of whiskers, giving an expression of surprise and fright. Section leading to the converse, with slight flushing of the upper gum. As, according to Dr. Hall White, the collateral ganglia of the sympathetic are atrophied and degenerate organs like the coccyx and appendix cæci, the question suggests itself, whether loss of hair from the front of the human scalp, the common form of baldness, is not a phenomenon due perhaps to degeneration of the superior cervical ganglion in man, contended for by White.

As to the action of *pilo-motor* nerves, Langley and Sherrington state (*Journ. of Physiol.*, 1891, p. 278) that the hairs of the monkey, cat, and probably all animals in which horripilation takes place under the influence of strong emotion, may be erected by stimulation of nerve fibres coming from the spinal cord and passing through the sympathetic system. The effects are mainly unilateral, but bilateral in the tail.

Langendorf (*Centralb. f. Physiol.*, 1891, p. 129) confirms the researches of Langley and Dickinson showing that the fibres of the sympathetic chain do not simply pass through the ganglia intercalated in their course, but end there, in order, so to speak, to be regenerated and to issue from them in a new state. Dastre (Compt. rend. Soc. de Biol.) comments on Langendorf's experiments, and claims that his researches on the same point and leading to similar conclusions were made in 1879–1884, and that the authors mentioned only confirmed his conclusions. That he, however, went beyond them and reached the general conclusion that the ganglia of the sympathetic chain are tonic and inhibitory centres. He holds that the vaso-constrictor nerves issue from them, while the vaso-dilators end in them, and claims analogous functional activity for the minute peripheral ganglia disseminated in the various organs.

Any consideration of the functions of the sympathetic system, at once brings forward the vexed question of *trophic nerves*, which finds an able treatment at the hands of Arndt (*Arch. f. Anat. und Physiol.*, 1891, p. 54 *et seq.*) in an exhaustive consideration of the subject from all points of view. The author holds that the existence of trophic nerves is

[1] Monitore Zoologico Italiano.
[2] Jour. of Physiol. 1891, p. 5.

beyond all doubt, and in support of this adduces experimental and clinical evidence. The latter, collated from a wide range of pathological observation, includes among many others, cases of paralysis with decubitus; surgical section of nerves with decubitus, lesions of the brain-stem followed by ulceration and perforation of the stomach and small intestine; jaundice after strong emotions, ascribed to trophic processes in the liver; desquamative nephritis from emotions; zoster, herpes, urticaria, due to trophic nerve influences. He holds that there must be trophic nerves, that this is proven by the numerous observations of Virchow, Nasse, Nélaton, Cl. Bernard, Légros, Onimus, Schiff, Joseph, Heidenhain and many others. Now, the question is, where are these nerves to be looked for, and how can we find them? As yet they have only been demonstrated directly on the corneal cells (Kühne), the pigment cells of amphibia and reptiles (Ehrmann), and on the cells enclosing the stomata of lymph spaces (Hoffmann), while their presence elsewhere has been assumed from physiological, but more especially from pathological data. They have been postulated, but also quite as frequently rejected, because not visibly demonstrated. This failure of demonstration is due to the fact that we had no clear idea as to what was to be looked for; and because we failed to recognize in its true character something we had long possessed, viz.: trophic nerves, that may be demonstrated at all times. To support this dictum, the author refers to his studies in 1890, proving that the nerves and the entire nervous system, developed as they are, from the ecto- and entoderm, represent a reflex rather than an automatic apparatus. Thus a given nerve is excited, *i. e.*, some process of movement passes along it and calls forth a corresponding movement. This movement passes from the place where it originated, to the end-cells supplied by the nerve, as is shown by production of heat, electricity, etc. The first thing, therefore, that is excited, or inhibited or destroyed by nerve action is metabolism, and as all the phenomena of nutrition depend upon this, it follows that the trophic processes must be influenced by any nerve excitation. Thus every nerve, and especially every centrifugal nerve, is primarily a trophic nerve, which, exciting trophic processes, leads secondarily to secretion, contraction, motion, usually supposed to be produced directly. These visible results are in reality only the outcome of trophic processes produced by the nerve. Afferent nerves act in a manner analogous to efferent nerves, by regulating the vital processes in the cells of the central nervous system, and the phenomena of sensation, consciousness, volition, etc., are the secondary but not the primary results of the impulses reaching the central cells by way of centripetal nerve fibres. Hence, according to the author, there is no necessity whatever to assume the existence of special trophic nerves, nor to ascribe trophic action to the sympathetic, when no other trophic nerves can be demonstrated. The sympathetic as a matter of course regulates the nutritive supply by its action on the vascular system, but it is trophic in the strict sense, only or especially for the smooth muscles supplied by it. Every nerve being trophic, should we retain the terms sensory, motor or secretory to designate the functional differences of nerves? For the sake of convenience, why not? But no nerve *per se* is either sensory, motor or secretory; it only causes sensation, motion or secretion by exciting its end-cell. It would therefore be better to speak of nerves as centripetal and centrifugal; or still better, to classify them as receptive and reactive; for upon reception of excitation and reaction thereto, brought about by chemico-physical processes, comprised under the more concise term of nutritive processes, all life is based, and this life finally is only a chemico-physical process, composed of what we briefly call assimilation, and in a wider sense of the term, secretion.

As the existence of special trophic nerves has been the favorite bone of contention among physiologists, while pathologists and clinicians appear long since to have reached an agreement from clinical phenomena, Arndt, in taking the broad ground that all nerves are trophic in the proper sense of the term, and that the evidences of their action as expressed in sensation, motion, secretion, etc., are but the secondary, the necessary outcome of their primary trophic function, propounds a theory that seems to embrace the greatest number of facts in a very satisfactory manner. As a "working theory" it appeals alike to the physiologist and the clinician, and offers a rational explanation of experimental as well as pathological phenomena, until now ascribed to the action or non-action of specifically trophic nerves. It certainly is a great and important step in the direction of removing this *quæstio vexato* of *trophic* nerves from the arena of physiological controversy.

The important field of reflexes has received some very valuable additions, representing perhaps the most positive gains made during the year, and certain to prove of great service in the diagnosis of a number of diseases of the nervous system.

Hughes (*Alienist and Neurol.*, 1891, p. 44) calls attention to a reflex (Onanoff's bulbo-cavernous reflex) possessing great physiological and pathological significance, because active in all healthy male adults with normal spinal cords, but absent in infants, feeble or absent in males before puberty. It is impaired by excessive venery, by masturbation, lowered in the later stages of typhoid fever. This reflex he calls the virile reflex, or penis percussion reflex. Its presence or absence is a valuable sign of the vigor, impairment, loss or abeyance of sexual power in the male. In a paper read before the Section of Medical Jurisprudence (JOURN. AM. MED. ASSOC., xvii, p. 727) he adds clinical evidence proving the value and significance of this reflex in diagnosis.

Jacoby (*New Yorker Med. Monatschr.*, 1891, p. 363) describes a new and interesting reflex phenomenon, produced by applying the galvanic current to the dorsal part of the hand and wrist. It consists in a short lightning-like contraction of the levator menti, when the current is closed; the contraction sometimes extends to the quadratus (both supplied by the facial nerve). In strongly marked cases, when both forearms are excited simultaneously by cathode, there is elevation of the chin, and the middle of the lip is protruded, giving to the face an expression of haughtiness or contempt.

Another addition to our knowledge of reflexes is made by Lazarus (*Arch. f. Anat. und Physiol.*, 1891, p. 19), who shows experimentally, that a reflex may be produced from the nasal mucosa to the bronchi; that certain irritants applied to the former reflexly cause a diminution of the lumen of the bronchi; and that the centrifugal part of this reflex is to be found in the vagus; also that it is more than probable,

almost certain, that this lessening of the lumen is due to contraction of the bronchial muscles. Zuntz, in speaking of this reflex, points to its bearing on the respiratory process. He says, it might seem that the greatest possible width of the bronchi would be most advantageous for respiration, and that in fact, contraction of the bronchi cannot be favorable to the filling and emptying of the lungs, but it possesses an essential significance in that it frees the air from all dust particles before it reaches the alveoli. The task to free the respired air from dust will be the more difficult, the greater the amount of dust contained in it. Hence a contraction of the bronchi becomes of importance, when the respiratory air is thus contaminated. This, indeed, renders respiration more difficult, but at the same time insures pure air to the alveoli. At the hand of these experiments, we can readily understand why in a pure air, free from particles of dust, we can so readily expand the chest, and why, *per contra*, when the respired air excites the nasal mucosa by dust, by its temperature, or by carrying irritant gases, the phenomena of labored respiration make their appearance.

A new reflex, *the anal*, is described by Rossolimo (*Neurol. Centralbl.*, 1891, 257). After speaking of the segmentary arrangement of reflex function of the spinal cord and its great importance in the localization of diseases of the cord, he deems a study of a new reflex segment not superfluous on anatomical, physiological and clinical grounds. The phenomenon in question occurs upon every contact with the integument and mucosa of the anus, and is accompanied by contraction of the sphincter ani, found in all normal men without exception. The integument of the anus and its mucosa as well as the sphincter ani externus are innervated by the external hæmorrhoidal nerve, formed from the third and fourth root of the sacral plexus. Hence, we may, upon these facts assume that the center of the anal reflex is near the third and fourth sacral roots, *i. e.*, in the lower conus medullas. In the dog it is located in that part of the cord corresponding to the third sacral root, *i. e.*, in the third fourth, counting from above, of the lumbar enlargement. The reflex arc is composed of the fourth sacral nerve and the centre, which in the cord lies above the root of the fourth, near the third pair of sacral nerves. It is thus placed below all other skin reflexes, even below the plantar reflex. It is pathologically increased: in neurasthenia, with increase of all skin reflexes, in high transverse myelitis, in anatomical disease of the nervous system with increased function of the sensory apparatus. It is decreased or destroyed in multiple neuritis with extension of the process to the sacral plexus; in cases of tabes, where there is disease of the pelvic organs and a more or less pronounced anæsthesia of the anal region; in myelitis of the lower segment of the lumbar enlargement. It remains normal in functional neuroses of micturition, defecation and sexual power. A knowledge of this reflex thus facilitates on the one hand the differential diagnosis of functional and organic disturbances of the pelvic viscera, and on the other a more accurate localization of the pathological processes in the spinal cord.

A curious form of what he calls inhibition, is described by Brown-Séquard (*Arch. de Physiol. norm. et pathol.*, 1891, p. 507). He thinks that surgeons might avail themselves of it, should the same conditions obtain in man, as found experimentally in the ape and dog. His experiments on these animals show that wounds produced by cutting or burning, may, after excitation of the larynx or its sensory nerves, become analgesic, and remain in that condition for several days, and even two weeks or more. He finds that wounds inflicted either before, during or a short time after certain irritations of the laryngeal mucosa or its sensory nerves, always present a total loss, or else a greater or less diminution of sensibility to painful impression.

The same author (*loco. citat.*, p. 805) supplements the foregoing by experimental evidence showing that in the ape and dog, a general reflex analgesia may be produced by a traumatic or mechanical irritation of the integument of the neck, the trachea, and especially of the larynx or the superior laryngeal nerves, and also by irritation of the laryngeal mucosa by chloroform, cocaine, etc. Should these results be verified in man, surgeons would be placed in possession of a ready and safe method for the production of local analgesia.

Wedensky (*Compt. rend. Acad. des Sciences*, 1891, p. 805), from a series of experiments undertaken with a view to solve the question in which part of the neuro-muscular apparatus inhibition is produced, is led to the conclusion, that the peripheral nerve endings, and not the muscular fibres pass into a state of inhibition, when frequent and strong excitations are applied to the neuro-muscular apparatus.

The physiology of the medulla oblongata has received attention at the hands of Spencer (*Proc. Royal Soc.*) whose researches were for the purpose of connecting more closely clinical signs with pathological changes in the medulla oblongata by localizing in the floor of the fourth ventricle the centers influencing respiration and circulation. He finds that the *respiratory* center lies along the middle line, extending for 2 mm. on either side, while that for *expiration* lies along the lateral part of the ventricle 2 to 3 mm. from the middle line. Upon the other hand, respiratory rhythm is slowed by excitation of an area lying over the continuation of the postero-median column, as the latter separates from its fellow on the opposite side, and that part of the floor of the ventricle close to the inner border of the column. Its central part is between 1 and 2 mm. from the column and between 2 and 3 mm. from the middle line.

Cardio-inhibition, although it may be produced all over the floor of the fourth ventricle, and also in rear of the calamus, is best marked over the posterior third of the ventricle, and over the inner margin of the continuation forward of the postero-median column. The chief depressor center is located in the hinder part of the floor between 1 and 4 mm. in front of the calamus, while rise of blood pressure is most frequent upon excitation of the floor from 4 mm. in front of the calamus to the anterior border of the ventricle.

The question of the existence or non-existence of a special center for vomiting, a long disputed point in the physiology of the spinal axis, appears at last to have been successfully solved by Thomas (*Arch. f. path. Anat.*, 1891, p. 49). At any rate, the experiments seem to leave no room for doubt that such a center exists in the dog and cat. He demonstrates that there is an independent vomiting center, as held by Mayer; that this central apparatus occupies a space 5 mm. long by 2 mm. wide, lying in front and rear of the calamus scriptorrius and within the deeper

parts of the medulla oblongata. It is functionally and anatomically independent, and represents a mechanism of a higher order connected with numerous other centers of a subordinate character, and capable through excitation of the latter, to produce its characteristic end effects. This, if correct, removes the question of a vomiting center from the region of controversy and hypothesis, where in spite of numerous investigations, it has remained heretofore, and thus marks a distinct advance in our knowledge of the functions of the medulla oblongata. It also disposes of Hlasko's theory (*Inaug. Dissert, Dorpat*), that the corpora quadrigemina are the centers from which the impulses causing contraction of the cardia proceed by way of the vagi and cord.

Ott (*Jour. of Nerv. and Ment. Dis.*) adds to our very meagre knowledge of the functions of the thalamus opticus, some experimental data pointing to its vasomotor influence, since section immediately in front of the pons, causes a fall of tension in the blood vessels. He thinks it quite probable that certain peripheral nerves stand in reflex relation to vasotonic centers in the thalami, which latter exert a vasotonic action upon the medullary dominant vasomotor center. These experiments throw some light on the obscure relations of the base of the brain to the chief vaso-motor center.

As to brain physiology, some important observations must be noted. Danilewsky (*Centralbl. f. Physiol.*, 1891, p. 1–4) adds to our knowledge of the electro-motor processes in the cerebrum, experimental evidence, proving that the conditions of excitation produced there by external irritants, are accompanied by definite electro-motor phenomena, the latter being regarded as objective signs of psychical processes, that subjectively appear as sensations, motions, hallucinations, etc. Thus, even feeble stimulation of a sense organ or of the internal sensory nerve (the vagus) produces an evident change in the electro-motor conditions of a definite region of the cortex, usually on the opposite side, which is set aside by deep morphia or chloroform narcosis. The *electro-motor* method of investigation is of value, not only in the question cerebral localization, but also in the investigation of the processes of excitation as such in the cerebral cortex.

Ireland (*Brit. Med. Jour.*, 1891, p. 1167) discusses the discordant action of the double brain, and shows that, one hemisphere remaining healthy, the other being diseased, the integrity of the intellectual, moral and emotional faculties may be preserved, and perhaps better preserved with a total destruction of one hemisphere, than with a diseased hemisphere transmitting irritation to its fellow.

Some interesting points on the cerebral circulation have been brought out by de Boeck and Verhoogen (*Institut. Solvay*) by studying some of the conditions under which it may be altered. They reach the following highly practical conclusions: In asphyxia the amount of blood in the brain is increased throughout, not because of increase of general blood-pressure, but from active dilatation of the cerebral vessels. Injection of sulphuric ether produces increase of arterial blood pressure, leading to a more abundant blood supply, while intravenous injection of morphia does not alter the total amount of blood in the brain, but changes the manner of its distribution, inasmuch as the cortex becomes anæmic, while the base is rendered hyperæmic.

Cybulsky (*Centralbl. f. Physiol.*, 1891, p. 834) holds that v. Bergmann's investigations on brain pressure and his theory thereon, afford a sufficient explanation of the subject, fully corresponding to clinical observation. Surgeons must be surprised to learn that Adamkiewicz looks upon brain pressure as impossible, even asserting that when local pressure is produced by inserting pieces of laminaria into the cranial cavity, there results, not anæmia, but hyperæmia. Cybulsky's investigations lead him to conclude that intra-cranial pressure is always positive, presenting considerable variations due to the state of the cerebral circulation. In dogs the variation is between 72 mm. to 190 mm. $H_2O$. Intra-cranial pressure, *i. e.*, pressure of the cerebro-spinal fluid, may be greatly increased by diminishing the cranial space by foreign bodies, *e. g.* a hæmorrhage from an artery outside of the dura mater. If under a certain degree of blood pressure, a 0.6 per cent. solution of sodium chloride, or oil be introduced beneath the dura, or if they, or an india-rubber bladder be forced between dura and bone, we see, when pressure is equal to that of the blood in the carotids, the symptoms of increased brain pressure, slow pulse, increased arterial pressure, acceleration, followed by slowing and finally cessation of respiration, loss of consciousness and death, with coma and tetanus. The rapidity of flow in the internal carotids is decreased as soon as intra-cranial pressure is increased; and again accelerated when with the same intracranial pressure, general blood pressure is increased. Whenever intracranial pressure becomes higher than the arterial, there is cessation of the cerebral circulation. These changes in the rapidity of the flow show that increased intra-cranial pressure leads to brain anæmia, and the symptoms mentioned are the results of this anæmia. (This fully agrees with v. Bergmann.) Injection of neutral or ammoniacal salt solution into the cerebral vessels does not produce these results. (This contradicts Adamkiewicz). He produced local pressure in rabbits by placing pieces of laminaria within the skull, and is convinced that this depression of brain substance is connected with displacement and disfiguration of the brain, and also with pressure on the blood vessels and ventricles. Such pressure remains latent only, when the volume of laminaria introduced does not exceed $\frac{1}{19}$ to $\frac{1}{20}$ of the volume of brain. If greater, there are changes in the circulation, numerous extravasations, producing death sooner or later. Adamkiewicz' assertion that the skull space may be decreased by $\frac{1}{8}$ or $\frac{1}{4}$, without production of death, depends as he thinks, on erroneous measurements.

That essentially modern offspring of our science, dating its birth from the discoveries of Fritsch and Hitzig (1870), and truly inaugurating a new era in the physiology and pathology of the brain, viz.: the doctrine of cerebral localization, or if you please, "scientific phrenology," has not fallen behind in the general advance of nerve physiology. Its importance *per se*, as a new physiological doctrine, and the brilliant results of its application in brain surgery, have made its study not only of great inherent interest, but of the highest practical value. While indeed, accepted by the majority of physiologists, it still finds able opponents in our ranks, and the literature of the past year shows that it yet lacks the seal of unanimous approval.

Perhaps the most important work on localization

published during the year is presented by Gotch and Horsly (*Philosoph. Trans.*, 1892, p. 269–526) being a magnificent monograph entitled "The Mammalian Nervous System, its Functions and their Localization Determined by an Electrical Method." It is the substance of the Croonian lecture, and after a full description of the method and plan pursued by the authors, it gives a full account of the important results of their investigations. It is impossible, within the limits of this paper, to do even scant justice to such a work by attempting to summarize its contents. It can only be fully appreciated by careful study, which, however, will fully repay for the time and labor employed by imparting information absolutely essential to a clear understanding of modern nerve physiology and its methods.

Beevor and Horsley (*Philos. Trans.*, 1891) in another finely illustrated monograph, give an extensive account of their experimental investigation of the motor paths in the internal capsule of the dog's brain. They show that the arrangement of the motor fibre corresponds exactly to the arrangement of the motor areas in the cortex. They also disprove that the trunk muscles, and those of the tongue of each side possess bilateral innervation, as heretofore supposed.

Donaldson (*Am. Jour. of Psych.*, 1891, p. 113) publishes an abstract of six lectures on cerebral localization, aiming especially to show the bearing of recent anatomical investigations on this point. After a description of Golgi's method of investigation of nerve structures, he gives a resumé of the advances in our knowledge of nerve cells and fibres and their mutual relation, presenting an excellent picture of the architecture of the cerebral nervous system. He next considers motor and sensory localization in man and ape, and discusses the results of experiments in animals lower in the scale, concluding with a consideration of the connection between localization and psychology.

These lectures form a valuable addendum to physiological literature, and the author deserves our thanks for the clear and concise presentation of the question in all its bearings.

Santmeyer (*Zeitsch. f. Biolog.*, 1891, p. 177) adds to our knowledge of conduction paths, by a description of the secondary degeneration following extirpation of motor centers in the dog. He finds that unilateral extirpation of motor centers frequently, but not always, leads to bilateral degeneration, and that the degeneration on the side of the lesion may occur as early as that in the opposite tracts. The degeneration on the same side has not as yet received a satisfactory explanation. The degenerated fibres appearing in the later stages in the anterior and posterior column, are not in direct connection with the extirpated centers. There are no anterior pyramidal tracts in the dog. The lemniscus, the nuclei of Goll's and Burdach's columns, the anterior spinal roots and the peripheral nerves are not attacked by secondary degeneration.

Bernheim (*Rév. Méd. de l ést.*, 1891, p. 513) illustrates motor localization in the cerebral cortex by a case of left brachio-facial monoplegia, followed by an attack of apoplexy with right hemiplegia. History and autopsy confirm the localization of motor centers in man as given by Charcot and Pitres. Brachio-facial monoplegia coincides with lesion of the inferior half of the ascending frontal convolution. 2. Sacral and lingual monoplegia depends on lesion of the inferior extremity of the motor zone, especially of the ascending frontal. 3. Brachial monoplegia on a limited lesion of the middle motor zone, especially of the ascending frontal. 4. Crural monoplegia on a very limited lesion of the paracentral lobule.

Shaw (*Brit. Med. Jour.*, 1891, p. 946) had the somewhat rare opportunity of examining the brain in a case of an old amputation of the thigh, and found atrophy of the cerebral cortex in the region corresponding to the leg center, with absence of the giant pyamidal cells of Betz from the ganglionic layer of the atrophied region.

While it is generally held that the auditory center is located in the first, or in the first and second temporal convolution, a view, however, not accepted by all, the report of a case bearing upon this point by Mills (*Brain*, 1891, p. 465) with history and autopsy becomes of great value. His conclusions from a study of the case are, that the center for word hearing is situated in the posterior thirds of the first and second temporal gyri, just in front of, or in a line with the posterior extremity of the horizontal branch of the Sylvian fissure. A lesion, confined to this region of the left hemisphere, produces complete, or almost complete word deafness. The field for all auditory memories is much larger, including at least, the posterior two-thirds of the first and second temporal. While these centers are most highly developed in the left hemisphere, destruction of the centers of both sides is necessary to produce complete brain deafness. The other temporal convolutions are not concerned in central audition. A lesion limited to the center for word hearing, causing word deafness, also causes paraphasia and paralexia, but it does not necessarily cause inability to recall words by other means, for instance, through their visual signs; in such cases probably the meaning of the word is understood, although the name cannot be properly verified in consciousness. Lesions causing word deafness will eventually lead to secondary atrophy of the speech and oro-lingual centers on the motor side of the brain, and to atrophy of the association tracts between the sensory and motor hearing-speech centers. The retro-insular convolutions are closely related with subdivisions of the first temporal gyrus, the most posterior being continuous with the posterior two-thirds of the first temporal gyrus.

The existence and localization of *cerebral* heat centers, first demonstrated by Landois and Eulenburg, has led to renewed investigations, notably so by White and Ott. White (*Jour. of Physiol.*, 1891, p. 271) discussing the position and value of those lesions of the brain, causing rise of temperature, states that after injury to the optic thalamus, produced by a blunt probe, the rise of temperature is slight as compared with that of a similar injury to the corpus striatum, while injury to the septum pellucidum causes a marked rise. Injury to the cerebellum produces no rise, while injury to the posterior part of the cerebral cortex has an effect more marked than injury to the frontal portion. There is a marked rise from injury to the crus cerebri.

Ott (*Journ. of Nerv. and Mental Diseases*,, 1891, p. 483) finds that the tuber cinereum is the centre for polypnœa and thermotaxis, and on the basis of his own researches revises the function of the thermotaxic centres as follows: In the fore-brain, the cortex contains thermo-inhibitory centres at the sulcus

cruciatus and the Sylvian fissure. Thermogenic centres are found at the base in the caudate nucleus, the gray matter of the septum lucidum, and the gray matter in front and beneath the caudate nucleus. The interbrain contains thermolytic, polypnœic and vaso-motor centers in the tuber cinereum; the afterbrain: thermolytic, respiratory and vaso-motor centres; the spinal cord: thermolytic, sudorific and thermogenic centres.

The localization of sensory centres is still a subject upon which opinions are widely apart, as may readily be seen by the following:

Saville (*Brain*, 1891, p. 270) concludes from a highly interesting case of anæsthesia with trophic changes in which the lesion was found to be strictly localized, as shown by the autopsy, being precisely beneath those parts of the cortex corresponding to the gyrus fornicatus and part of the marginal convolution, in their entire extent, and beneath the anterior half of the præcuneus; cutting off all direct communication between the gyrus fornicatus and the white matter beneath, and in front between the gyrus fornicatus and the marginal convolution and centrum ovale, that the gyrus fornicatus is the centre for common and tactile sensations, and that its destruction may produce loss of sensation without loss of motion to any serious extent. (This case, therefore, is in line with the conclusions arrived at by Horsley and Schäfer.) He also concludes, that a destructive lesion in that portion leads to vaso-motor or trophic changes in the skin and subcutaneous tissues of the opposite side of the body, and that therefore these convolutions may possibly be the centre, not only for sensation, but for trophic influences transmitted to the opposite side of the body.

*Per contra* Knapp (*Boston Med. and Surg. Journal*, 1891, p. 430) presents the details of a case, tending to show that the centres for sensation of touch, pressure, motion and position are to be looked for in the central convolutions. The lesion involved the middle third of the left ascending parietal convolution. Sensations of pain and of temperature were not abolished. The limbic lobe was not involved in this case, as it presented no signs of inflammation. These results accord singularly with those of Munk's experiments, although Knapp does not fully accept Munk's opinion, that the cerebral cortical region is purely sensory.

Schtscherbach (*Centralbl. f. Physiol.*, 1891, p. 289) attempts to solve the difficult question of localization of the centre of taste in the rabbit, and finds that injury to an area extending from 2 to 3 mm. in front and behind the line of the coronal suture and vertically from the longitudinal fissure to the lower edge of the brain, causes loss of *taste* in the opposite side of the tongue, lasting for six days at most.

As opposed to the doctrine of localization, we find Lane (*Am. Journ. of Insanity*, 1891, p. 50), who holds, speaking of the so-called motor area of the cortex, that the theory of cerebral localization is not supported by sufficient evidence, and therefore concludes that, upon the facts presented, it is simpler and more consistent, therefore wiser, to consider the cortex (so far as explored) as an entirely sensory region, the seat of memory and will.

While Bennett (*Dubl. Journ. Med. Sci.*, 1891, p. 337) records the history of a case of cerebral tumor, illustrating the difficulties of cerebral localization. The tumor had destroyed the centres of volition governing the left arm and also those of other parts of the left side of the body, obliterating the lower two-thirds of the fissure of Rolando, and involving nearly all of the lower half of the ascending parietal; nearly the entire ascending frontal convolution was lost in the tumor, yet never during life was there any paralysis of the limbs. He asks, how are such facts to be reconciled with the present views of cerebral localization?

We finally must consider what has been done in advancing the physiology of the cerebellum, that fruitful field for speculation, to which numerous hypotheses have assigned at some time or other the *rôle* of being the seat of nearly every functional manifestation of the central nervous system, and which even now, in spite of its ready accessibility, of countless experiments, of an immense amount of clinical observation, and of the fact that some of our most skilled and assiduous investigators have made it their special object of research, is an organ whose physiological significance is enveloped by doubt and uncertainty. For we must confess that we have gained but little positive knowledge since the brilliant experiments of Flourens opened up the question of cerebellar function, and pointed to its connection with the motor apparatus. Whether the latest contributions have brought us much nearer to a solution of this perplexing question may perhaps, and not without reason, be doubted, nevertheless the two most striking and exhaustive monographs dealing with the physiology of the central nervous system published during the year, and presently to be considered somewhat in detail, aim to offer a solution of the question of cerebellar function as a whole, and their authors endeavor to establish theories, which although widely apart, are well presented, clearly formulated and supported by ingenious arguments, that may or may not be deemed convincing or acceptable to physiologists. Before touching upon these, however, it will not be amiss to make brief mention of a few of the experimental data on cerebellar function, that have been published during the year. Thus:

Borgherini (*Neurolog. Centralbl.*, 1891, p. 649) describes a novel phenomenon, consisting in a sleep-like state induced in dogs after removal of the cerebellum, by bandaging the eyes with a thick cloth. In this condition the animal does not respond to any mechanical or tactile excitation. The author believes that the absence of visual impressions produced by the bandage causes in the animal a consciousness of complete inability to move, and forces it into absolute immobility; it has lost the will to move.

Lange (*Pflüger's Arch.*, 1891, p. 615) shows experimentally that the symptoms due to ablation of the cerebellum must be strictly separated from those after destruction of the semicircular canals, thus opposing the views of Baginsky, who regards the auditory symptoms in the light of brain disturbances, and of Loeb, who has lately again supported the older view, that the cerebellar symptoms may be referred altogether or in greater part to lesion of the auditory nerve.

We now pass to a consideration of the two monographs that represent the most noteworthy additions to the literature on the cerebellum and its functions which have made their appearance for several years past. The first of these, by Luciani, is a volume of some 300 pages, entitled "Il Cerveletto," of which

Pescarolo (*Arch. Ital. de Biol.*, xvi, p. 289) gives a very full and clear abstract. The author presents a series of new experimental studies on the normal and pathological physiology of the cerebellum, with new facts that, according to Luciani, demonstrate the error of the doctrines now held, and tending to assist the physiologist and clinician in the interpretation of certain phenomena. The fundamental fact derived from these researches and from the best clinical and experimental observations is, that absence of a more or less extensive or deep portion of the cerebellum, or its total absence even, is not followed by any paralysis, partial or total, of either sensation or motion, or by disturbance of the intellect or volition. This leads him to look on the cerebellum, with all its dependencies, as a small system, possessing a comparative autonomy, and to a certain point, distinct from the great cerebro-spinal system. It is not an intermediate organ, intercalated along the great tract of cerebro-spinal transmission, but rather an appendicular organ, which, by means of different afferent routes, is directly or indirectly *en rapport* with the peripheral sense-organs, and by special efferent routes has a direct connection with certain collections of gray matter of the cerebro-spinal axis, and indirect connection with the peripheral apparatus of voluntary movements. It, like the rest of the cerebral nervous system, is an organ with bilateral action, but mostly direct, unlike the mainly crossed action of the cerebral hemispheres. Its influence is not limited to the muscles of station and locomotion, but extends to all voluntary muscles, though not in the same degree, its action being more pronounced on the muscles of the lower limbs and those fixing the spine. The cerebellum is an homogeneous organ as to function; *i.e.*, every segment has the function of the whole, and in absence of the rest may supplement them, provided the normal relations to the afferent or efferent tracts be not altered.

As absence of the cerebellum, whether in animals or man, is not followed by paralysis, or imperfection or lowering of the external or internal sense, it is believed that it does not possess sensory function; still, considering the inherent imperfection in the methods employed to examine the different sensations in animals, and the difficulties of the subjective examination of the sick, because of the ready *suggestionabilité* of the subjects, the author abstains from pronouncing on this question, and leaves it to future investigations. The grand total of results shows, that ablation of the cerebellum is always followed by neuro-muscular phenomena of an asthenic, atonic and astatic character, hence the influence of this organ upon the rest of the system is complex in character, procuring, 1, an increase of potential energy at the disposal of the neuro-muscular apparatus (sthenic action); 2, an increase of their tension during functional pauses (tonic action); 3, an increase of the rhythm of elementary impulses during action, with a normal fusion and regular continuity of the act (static action). And as every functional excitation of living elements is necessarily accompanied by a modification of nutritive movements it follows, that the complex action of the cerebellum is connected with a trophic action directly or indirectly. This trophic influence is naturally slow, tranquil, continuous, yet varied in intensity in the normal condition of the organism, and the functional influence of the cerebellum, normally exerted upon the other nerve centres, is likewise slow and continuous. The activity of the cerebellum is not specific, or *sui generis;* it is the common, fundamental activity of the entire nervous system.

The peripheral nerve ganglia of the sympathetic system, possess fundamentally an analogous relationship to the functions of vegetative life, as demonstrated by Luciani's researches on the functions of the ganglia of the posterior roots, which are claimed to have shown, that, like the cerebellum they possess a trophic action and also the function of reinforcement, both to be considered as different manifestation of the same process, that constitutes the unexplored and as yet mysterious field not only of the physiology of the cerebellum and of the intervertebral ganglia, but of that of the nervous system in general.

The other work referred to, differs from the foregoing, being of a decidedly revolutionary character, as the views expressed by its author are opposed to all that is held to be almost axiomatic in cerebellar physiology, the doctrine, that this organ does not take part in the mental processes. This volume of some 600 pages is by Courmont, and bears the title: "Le Cervelet et ses Functions, Paris, 1891." It received what may be called a preliminary review at the hands of Prof. Forel (*Bull. Med. du Nord.*, 1891, p. 244), from whose article the following synopsis has been obtained. According to Ford, the author, one day, while sitting at the bedside of a patient, was seized by an original and bold idea of cerebral physiology, which he formulated into an hypothesis of the functions of the cerebellum, according to which the latter is an organ *de sensibilité* and endowed with psychical functions. To verify this hypothesis, and judging that vivisection (although direct experiment was not neglected) because of the mutilations attending it, supplied less clear results in questions of cerebral localization, than those obtained from a study of clinical facts followed by autopsy; he searched, to use Forel's words, with the patience worthy of a benedictine monk (some might say, worthy of a better cause) through all the original French and foreign observations on cerebellar disease, and was astonished to find that they all confirmed his *á priori* conclusion. Again certain anatomical considerations, and experiments conceived in quite a different spirit and conducted with another end in view, equally supported the hypothesis. Comparative anatomy also supplied serious arguments in favor, and finally direct experiments brought all the confirmation they could offer to the author. Now, since the time of Flourens, the functions of the cerebellum are held to be those of coördination, but the author says, if setting aside all prejudice established by science, some one should propound to a physiologist the following: Given, an important encephalic organ, nearly identical in structure with the cerebrum, and not only placed alongside of but associated with it by close and intimate anatomical connections, should not such an organ possess functions analogous to those of the cerebrum? Clearly, the answer would be: *Yes.*

Now, the cerebrum is a motor organ, and at the same time that of thought, and Courmont holds, that the cerebellum also is the general receptacle for sensations and presides over a special class of psychic manifestations. Dividing the faculties into rational and emotional, he holds that reason, judgment, volition, etc., belong to the cerebrum, while the emotions,

love, fear, hatred, etc., have their seat in the cerebellum. (Jessen of Erfurt, formulated this idea in 1869, but advanced no proof in its support.) Courmont adduces in proof arguments from anatomy, based on analogy and held to establish a probability only; thus the cerebrum and cerebellum are each composed of two hemispheres, each contains a central white, and a peripheral gray substance. In both, the nerve fibres arise from nerve cells and form peduncles passing to the spinal cord. True, the cerebellum is much smaller but its gray substance is more abundant than the white. The comparative weight of the cerebellum is less in the male than the female, and in the latter the emotions predominate. Finally the motor columns of the cord pass to the cerebrum, the sensory to the cerebellum. And Kölliker is cited as holding that the anatomical disposition of the cerebellum illy accords with the theories of its motor functions.

As to experiment, Vulpian and Ferrier are cited, to prove that animals deprived of the cerebellum still experience pain and give signs of emotion. But clinical observations with autopsies supply the main foundation of the new idea, as a like observation first gave birth to it, and as this case shows more clearly the author's method of reaching conclusions, it may properly be briefly reproduced: A woman with puerperal mania presented a singular condition. She responded sensibly and rationally to all questions bearing on external facts, but at once became delirious when a word was pronounced, that set her emotional faculties into play. Now, rejecting the idea of two different classes of cells in the cerebrum, because the circulatory disturbance presumably producing the puerperal mania, could not affect one class, without at the same time acting upon the other, our author concludes, that there must be two organs, one presiding over reason, the other over emotion, and in his further search he finds divers psychical symptoms in cases of cerebellar lesion, such as changes of character, inexplicable irascibility, sadness without external cause, etc., claimed in support of the theory. As to experiment, he finds that ablation of the cerebellum in rats is followed by apathy, while its complete removal, producing inflammation of the remainder exaggerates the emotional conditions. The comparative and absolute highest development of the cerebellum in man is also held as proof in his favor. Other considerations are brought to bear; thus in examining the origins of the cranial nerves, he finds that those coming in part or altogether from the cerebellum, cerebro-peduncles or tubers, are the ones most affected in the emotions: the lachrymal, patheticus, facial, auditory. The anatomical disposition of the last affords an original explanation of the fact, that music, of all the arts, has the highest power to move man by means altogether exclusive of reason. Finally, the cerebellum, like the cerebrum, receives sensory impressions, as shown by the origin of the auditory nerve, the trigeminus, the posterior columns of the cord.

The psychical and sensory theory of the cerebellum admitted, our conception of the cerebral nervous system is according to Courmont, greatly simplified according to the following plan: there are two grand divisions, the spinal cord and the encephalon. Each is composed of two parts: the cerebrum and the anterior spinal cord are motor, the cerebellum and the posterior cord sensory, and upon these functions the two psychic functions, volition and emotion are based.

The motor disturbances from lesions of the cerebellum are explained by the supposition, that the posterior encephalon acts by a reflex mechanism upon the anterior encephalon, an action analogous to that of the posterior cord on the anterior; hence we see incoördination from experimental lesions of the cerebellum, and uncertainty of gait in cerebellar disease. Thus in a general point of view, the cerebellum becomes a highly important, but *indirect* factor in normal motility.

Such is a brief, and perhaps imperfect outline of the plea offered by Courmont in behalf of his theory of cerebellar functions, intended to take the place of what is held to be fairly well established by experimental researches and clinical observation, viz., that this organ is not connected with the phenomena of sensibility or thought, but only with those of coördination of motion, its influence being exerted especially upon those muscular actions that maintain the equilibrium of the body. That his theory is simplicity itself, goes without saying, that it is not altogether new, is shown by the author's reference to Jessen,[3] but it seems very doubtful, whether it will succeed in gaining acceptance in the place of what certainly seem to be logical conclusions as to cerebellar function, derived from generally admitted physiological and pathological facts. The author's work, however is a highly instructive example of the manner in which an enthusiastic and ingenious mind, bent upon establishing an *a priori* hypothesis in physiology, may find an abundance of material in every direction, that readily lends itself to supply the arguments in its favor.

There are many other points of interest connected with nerve physiology, that might well claim attention, especially those bearing on the special senses, but your kindly patience has already been taxed too much, to permit of any further trespass upon your time.

Summing up the progress made, not only in the physiology of the nervous system, but in all other fields of our science, we see in every direction a steady advance, a gradual, but no less certain and solid gain in our knowledge of the functional activity of the organism. And it is particularly gratifying, in fact a matter of just pride, to be able to note that the original investigations of American physiologists have added no little to this increase, the fruits of their scientific labors being fully equal in value to those of their foreign colleagues.

In conclusion, may we not express the hope, that now, with this new, independent Section of Physiology at their service, they may in future select its meetings and proceedings as the channel, through which they can most appropriately communicate the results of their observations and research to the scientific world?

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TO REMOVE NITRATE OF SILVER STAINS FROM THE FINGERS. — First paint the blackened parts with tincture of iodine; then, by applying ammonia, the iodine will be bleached, leaving white instead of black stains of nitrate of silver.

CANADA has had a single examining board for more than twenty years. During this period it has licensed 1,300 regulars and 19 homœopaths.

[3] See also Gowers, Diseas., of the Nerv. Sys., Amer. edition, p. 495.

## PURULENT BRAIN DEPOSITS, AND PHLEBITIS AND THROMBOSIS OF THE CEREBRAL VEINS AND SINUSES FOLLOWING EAR DISEASE.

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(*Continued from page 604.*)

*Case 135.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Male, age eight. Left ear. Chronic otorrhœa; vomiting; convulsions; paralysis of left upper eye-lid; limbs all weak; pain in left ear; dull; drowsy; semi-comatose; coma. Death.

*Autopsy.*—Abscess in left cerebral hemisphere.

*Case 136.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Female, age twenty-six. Right ear. Purulent otorrhœa; delirium; episthotonos; coma. Death.

*Autopsy.*—Abscess in under surface of middle cerebral lobe.

*Case 137.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Female, age fifty-one. Left ear. Purulent otorrhœa; cough; pain in limbs; pulse quick; convulsions; coma. Death.

*Autopsy.*—Abscess in left cerebral hemisphere.

*Case 138.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Female, age twenty-three. Left ear. Epilepsy; convulsions; pain in head; fever; intense agony; convulsions. Death.

*Autopsy.*—Abscess. Coagulum of fibrin and blood in left lateral sinus.

*Case 139.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Male, age fifty-four. Right ear. Epilepsy; pain in forehead; stupor; lost consciousness and sensibility; convulsions. Death.

*Autopsy.*—Abscess in middle lobe of right hemisphere.

*Case 140.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Female, age seven. Left ear. Purulent otorrhœa; great debility; epilepsy after syringing; epilepsy continued. Death.

*Autopsy.*—Abscess in under part of left lobe of the cerebellum.

*Case 141.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Male, age not given. Right ear. Purulent otorrhœa; cessation of discharge; chills and collapse; pain in right side; stupid; coma. Death.

*Autopsy.*—Abscess in right middle lobe.

*Case 142.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Female, age twenty-six. Right ear. Chronic otorrhœa; pain in ear; headache; dizziness; coma. Death.

*Autopsy.*—Abscess in upper part of right cerebral hemisphere.

*Case 143.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Female, age nine. Left ear. Purulent otorrhœa; fever; vomiting; pain in ear; paralysis of left side; coma. Death.

*Autopsy.*—Abscess in middle lobe of left side of cerebrum.

*Case 144.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Male, age thirty-two. Chronic otorrhœa; chills; fever; abscess behind ear; stupor; convulsions. Death.

*Autopsy.*—Abscess in middle cerebral lobe. Pus between diseased mastoid and dura-mater.

*Case 145.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Male, age thirty-five. Right ear. Chronic otorrhœa; caries of the mastoid; polypus in external meatus; pain in back of head, neck, and shoulders of the right side; stupor; coma. Death.

*Autopsy.*—Abscess in right lobe of cerebellnm.

*Case 146.*—Extract from Treatise on the Ear, by Roosa. Reported by Gull and Sutton. Male, age thirteen. Right ear. Purulent otorrhœa; fever; headaches; thick speech; hemiplegia; vomiting; drowsiness; pain; stupor. Death.

*Autopsy,* Three abscesses in right lobe of the cerebellum.

*Case 147.*—Extract from Treatise on the Ear, by Roosa. Treated by J. Orne Greene. Male, age twenty-eight. Right ear. Chronic otorrhœa; paralysis of the muscles of face after walk in rain; pain on right side of head; vertigo; chills; nausea; vomiting; coma. Death.

*Autopsy.*—Abscess in right half of the cerebellum. Roof of tympanum bare, but not carious.

*Case 148.*—Extract from Treatise on the Ear, by Roosa. Treated by J. Orne Greene. Male, age twenty-two. Right ear. Acute purulent otorrhœa; pain in ear; headache; difficulty in swallowing; vertigo; paralysis of right hypoglossal nerve.

*Autopsy.*—Meningitis. Caries of inner table of skull. Cochlea, and semi-circular canals filled with solid red mass.

*Case 149.*—Extract from Treatise on the Ear, by Roosa. Treated by Farwick. Female, age thirty-six. Left ear. Chronic otorrhœa; pain in ear and left side of head; vertigo; delirium. Death.

*Autopsy.*—Abscess in left cerebral hemisphere. Caries of the roof of tympanum.

*Case 150.*—Extract from Treatise on the Ear, by Roosa. Treated by Schwartze. Female, age eighteen. Left ear. Chronic otorrhœa; pain in ear; chills; region of left jugular sensitive; pain in swallowing; nausea; uvula œdematous; vomiting; singultus; left side of neck œdematous and painful on pressure. Slight convulsive movements of the left arm. Death.

*Autopsy.*—Clots in superior longitudinal and superior petrosal sinuses. Old thrombus in left lateral sinus.

*Case 151.*—Extract from Treatise on the Ear, by Roosa. Treated by Schwartze. Male, age three. Left ear. Purulent otorrhœa; meningitis. Death.

*Autopsy.*—Œdema of pia-mater. Left lateral sinus contained a thrombus. Carious bone in left auditory canal.

*Case 152.*—Extract from Treatise on the Ear, by Roosa. Treated by Schwartze. Female, age fifty-four. Right ear. Chronic otorrhœa; pain in ear and head; vomiting; vertigo; coma; ptosis of right side. Death.

*Autopsy.*—Hyperæmia of membranes of brain. Oedema of pia-mater. Thrombus in right superior petrosal sinus.

*Case 153.*—Extract from Treatise on the Ear, by Roosa. Treated by von Tröltsch. Male, age fifty-three. Left ear. Chronic otorrhœa; polypus; pain in ear and head; coma. Death.

*Autopsy.*—Abscess in left middle cerebral lobe, connecting with petrous bone.

TABULAR REPORT OF PURULENT BRAIN DEPOSITS, AND PHLEBITIS AND THROM-

| No. | Where Reported. | Surgeon's Name, etc. | Sex. | Age. | Ear. | Previous History. |
|---|---|---|---|---|---|---|
| 1 | Original | Allport, Minneapolis | F | 25 | L | Chronic otorrhœa for twenty-two years |
| 2 | Original | Allport, Minneapolis | M | 23 | R | Acute purulent otorrhœa |
| 3 | Original | Allport, Minneapolis | M | 22 | R | Chronic otorrhœa |
| 4 | Original | Allport, Minneapolis | M | 34 | R | Chronic otorrhœa, with acute exacerbations |
| 5 | Original | Allport, Minneapolis | M | 25 | R | Blow on ear, followed by acute purulent otorrhœa |
| 6 | Original | Allport, Minneapolis | M | 35 | L | Mental condition bad on entrance to hospital, thus obscuring previous history; chronic otorrhœa; pain in head |
| 7 | Arch. Otology, 1885 | Hedinger, Stuttgart | F | 63 | R | Chronic otorrhœa; fibrous tumor in meatus; numbness r. side of neck and face. Patient disappeared for years. On reappearance, had pain in ear; aural hæmorrhage streaked with pus; total deafness r. ear; pain in back of head; pain and swelling of temporal region |
| 8 | Arch. Otology, 1885 | Hedinger, Stuttgart | F | 50 | R | Chronic otorrhœa |
| 9 | Arch. Otology, 1886 | Truckenbrod | M | 28 | L | Chronic otorrhœa; vertigo |
| 10 | Lancet, 1885 | Hilles | | | | Chronic otorrhœa |
| 11 | Lancet, 1880 | Allen | F | 19 | R | Chronic otorrhœa; deafness |
| 12 | Gazette des Hopit., 1880 | Miot, Paris | M | 35 | R | Chronic otorrhœa; tubercular |
| 13 | Jour. Anat. and Physiology, vol. xiv | McBride & Bruce | | | R | Chronic otorrhœa |
| 14 | Arch. Otology, 1879 | Kipp | M | 23 | R | " " |
| 15 | Am. Otol. Society, 1882 | Mathewson, Brooklyn | M | 11 | | " " |
| 16 | Am. Otol. Society, 1882 | Merrill, Albany | M | 32 | | |
| 17 | Am. Jour. Med. Sciences, 1882 | Prentiss | M | 32 | R | Chronic otorrhœa |
| 18 | Am. Jour. of Otology, 1882 | Field, England | M | 42 | L | " " |
| 19 | Phila. Med. Times, 1881 | Harlan | F | 14 | L | " " l. facial paralysis |
| 20 | Edinburg Med. Jour., 1881 | Sinclair | M | 22 | R | " " caught cold |
| 21 | Australian Med. Jour., 1881 | Robertson | M | 39 | L | |
| 22 | New York Med. and Sur. Brief, 1879 | Moore | M | 50 | R | Chronic otorrhœa |
| 23 | Glasgow Med. and Sur. Jour., 1880 | Barr | M | 17 | L | " " |
| 24 | Jour. of Anat. and Phys., 1888 | McBride & Bruce | F | | R | " " |
| 25 | Gazette des Hopit., 1885 | Jaccoud | | | R | |
| 26 | Von Langenbeck's Arch., vol. 28 | Gluck | | | R | Chronic otorrhœa; aural hæmorrhages |
| 27 | Arch. Otology, 1880 | Michael, Hamburg | M | | L | Blow on head years ago; recovery ensued in a few days, with occasional pain on left side of head; discharge from both ears; deafness |
| 28 | Lancet, 1880 | Morris | M | 31 | L | Chronic otorrhœa; deafness |
| 29 | Arch. Otology, 1880 | Frankel | F | 23 | L | " " |

BOSIS OF THE CEREBRAL VEINS AND SINUSES FOLLOWING EAR DISEASE.

| Present History. | Result. | Autopsy. |
|---|---|---|
| Deafness; bony narrowing of meatus; pain in head; chills; temp. and pulse medium; slight delirium; mastoid opening; found pus | Death . | Necrosis found on outer surface of mastoid, squamous, frontal and sup. maxilla; on inner surface of squamous; on upper part of petrous; on petrous over middle and internal ears; on inner plate of mastoid. Pus found on outer surface of mastoid, squamous, frontal, and sup. maxilla. Pus generally distributed over left side of brain and first frontal fissure, first frontal convolution, fissure of Rolando, middle lobe on right side, both lateral ventricles, medulla and pons. Abscess on cerebellum. Abnormal openings existed through inner mastoid plate, through squamous, and between middle and internal ears and cranial cavity. |
| Pain in right side of head and ear; temp. and pulse medium; delirium; tender mastoid; mastoid opened; no pus found; squamous trephined; no pus found; skull trephined through mastoid and occipital bones; found pus | Death . | Abscess of cerebellum. Pus around medulla. Dura-mater inflamed and adherent. Pia-mater inflamed. Carious opening in mastoid plate (inner). Carious opening between middle and internal ears and cranial cavity. Thrombi found in longitudinal sinus, Sylvian fissure, sup. petrosal sinus and right ventricle. |
| Pain in right side of head; temp. and pulse medium; delirium; mastoid swelled and inflamed; mastoid opened; found pus | D'ath fr. ty'd fev. | Dura-mater congested and adherent. Pia-mater congested. Thrombi in longitudinal and lateral sinuses. |
| Pain in right side of head; temp. and pulse high; chills; delirium | Death . | Necrosis of tympanum and lateral sinus. Ossicles gone. Pus in sup. petrosal and lat. sinuses. Dura-mater and pia-mater inflamed. Thrombus in sup. petrosal sinus. |
| Temp. medium and sub-normal; pulse medium; pain in right ear, mastoid, and right side of head; mastoid swelled and tender; delirium; bowels loose; mastoid opened | Death . | Diffuse subdural abscess. Most marked in anterior parietal region, anterior to fissure of Rolando. Collections of pus on surface of r. frontal convolution, and r. temporal convolution. Purulent thrombus in the sup. sinus and in sup. longitudinal sinus. Adhesions between arachnoid and pia-mater. Carious opening from mastoid cells into lateral sinus. |
| Diarrhœa; chills; delirious; aphasia; incoherence; swelling under mastoid in neck; temp. and pulse medium | Death . | Large vessels of dura and pia-mater filled with dark blood. Basilary artery and circle of Willis in same condition. Inferior surface of cerebellum purulent. Middle and internal ears purulent. Purulent thromus in l. petrosal sinus, both sup. and inf. Same condition in lateral sinus and internal jugular vein. Purulent phlebitis of external jugular vein. The vein from auditory canal in the same condition. Pus in semi-circular canals. Thrombus of basilar vein. Superficial posterior auricular glands much enlarged. |
| Temp. and pulse medium; temporal swelling incised and pus liberated; cheek incised and pus liberated; mastoid muscle incised and pus liberated; mastoid opened and pus liberated; delirium; right pupil contracted; stupor; unconsciousness; spasms of l. upper and lower extremities; l. facial paresis | Death . | Multiple pus sinuses in neck. Carious spots on outside of mastoid and occipital. Congestion of dura and pia-maters. R. sigmoid sinus, bulbous portion of jugular vein, and mastoid emissory vein, all obliterated. Tumor in middle ear. Malleus and incus gone. Pus in mastoid antrum. Carious canal leads from mastoid antrum into sigmoid fossa. Carious opening through incisura Santorini. Caries of pyramid. Caries of bony wall of transverse sinus. |
| Vertigo; pain in head; marked in occipital region; temp. and pulse sub-normal; polypus in r. ear; removed; l. ear; chronic otorrhœa; simple; coma | Death . | Hyperæmia of all meningeal veins. Carious opening in sup. petrosal sinus over vestibule. Petrous bone carious. Semi-circular canal, vestibule, and cochlea carious. Pus in middle cranial fossa running into the canal of the medulla. Abscess in left posterior cranial fossa. Cholesteatoma in l. mastoid antrum. |
| Chills; painful spot on top of head; meatus narrowed; headaches; temp. medium; mastoid opened; found pus; paresis of r. side of face; difficult speech; defective memory; aphasia; diagnosis; brain abscess; operation; fistula directly above meatus enlarged; pus found | Recov'y | |
| | Death . | Cerebral meningitis. Abscess of l. temporal lobe. |
| Earache; head painful; vomiting; paraplegic; motor paralysis of lower limbs; scalp and spine painful to touch | " | Abscess beneath dura-mater on anterior surface of petrous bone. Drum-head and ossicles gone. Pus in middle ear, mastoid cells, and labyrinth. Pia-mater congested. Vertebræ in portions of cervical and dorsal spine carious. |
| R. facial paralysis. Painful mastoid | " | Pus in middle ear and mastoid cells. Ossicles gone. Caries of tympanic walls. Dura-mater red, thickened and softened. Facial nerve and chorda tympani largely destroyed. |
| | " | Pus in middle ear, mastoid cells, vestibule and cochlea. Abscess in outer half of r. cerebral hemisphere. |
| Acute exacerbation; pain over r. side of head; meatus red; chills, fever, vomiting, headache; optic nerves congested; convulsions, unconsciousness, coma | " | Longitudinal sinus filled with blood. Abscess in temporal lobe. Not encapsulated. Pus in lateral sinus. Opening in anterior and outer wall of lateral sinus. Thrombus in lateral sinus. Pus in tympanum and mastoid cells. Malleus and incus carious. |
| Mastoiditis; convulsions | " | Abscess of cerebellum. Pus between dura-mater and tegmen-tympani. |
| Acute otitis | " | Pus over the region of the petrous bone, extending from the tympanic cavity through openings in tegmen-tympani. |
| Earache and headache; chills; temp. high; pain in temporal region and over r. eye | " | Abscess in anterior lobe of l. cerebellum. Caries of petrous bone. Clotted blood in lateral sinus. Carious opening in wall of lateral sinus, communicated with abscess. |
| | " | Congestion of dura-mater. Puro-lymph in pia-mater over l. sphenoidal lobe. Abscess in l. lobe of cerebellum. Temporal bone necrosed, especially in tympanum. Pus in middle ear. Drum-head gone. |
| Unconscious; maniacal; r. pupil contracted; mastoid swelled; Wilde's incision | " | Meningitis. Abscess of l. lobe of cerebellum. Almost entire destruction of anterior wall of ex. meatus. Drum-head gone. Caries of middle ear. |
| | " | Abscess communicating with middle ear in tempero-sphenoidal lobe. |
| Deafness; purulent discharge from ear; l. facial paralysis | " | Caries in tympanum. Abscess in l. middle crus-cerebelli, extending partly into cerebellum and into pons, abutting on fourth ventricle and pressing on seventh nerve. |
| Acute exacerbation; mastoid abscess; opened; vomiting; semi-consciousness and facial paralysis; divergent strabismus; pupils contracted | " | Caries extending backward and upward from mastoid as far as parieto-occipital suture. Opening through parieto-occipital suture, leading into skull. Lateral sinus empty and hard. Meninges congested. Pus in posterior fossa between dura and skull. Caries at this point. Abscess of r. lobe of cerebellum. |
| Pain, especially in l. side of forehead; languid and drowsy; vomiting, aphasia, epilepsy | " | L. sphenoidal lobe adherent to bone. Abscess in l. sphenoidal lobe. Petrous carious in two places, one through roof of tympanum, the other in groove for lateral sinus. The latter communicated with mastoid cells. |
| | " | Abscess in r. lobe of cerebellum. |
| Pain and deafness; discharge; vomiting and dizziness; mastoid painful; fever | " | Pus at base of middle lobe of cerebrum. Tympanum purulent, granular, and carious. Drum-head gone. |
| Headache, fainting, convulsions, amaurosis, sopor, facial paralysis; paralysis of right arm; operation; posterior wall of meatus chiselled away; also portion of mastoid; dura exposed and fluctuating; pus between dura and pia | " | Purulent degeneration of dura, especially from longitudinal sinus to base of brain. |
| Headache, starting on l. side; feeling of pressure; stupid, aphasia; head drawn downward into neck; face red, pupils sluggish; constipation; polypi in both ears; removed; spasms; unconsciousness; flexion of all four extremities; delirium, stupor, sensitive skin, pain upon moving back | " | A yellowish projecting spot existed somewhat above surface of dura, over the crista sup. of the petrous bone, corresponding to site of injury. Pia, at convexity, covered with pus. Abscess in l. tempero-sphenoidal lobe. Pus in ventricle. An opening existed outward from the corpus striatum communicating with abscess. Purulent degeneration of temporal lobe. Frontal lobe œdematous and softened. Lateral ventricle as well as 3rd and 4th, filled with pus. Pia at base charged with pus, extending down into vertebral canal. Both mastoids filled with pus. |
| Pain; red mastoid; chills; unconsciousness; mastoid opened; pyæmia; herpes on face | Death . | Thrombi in l. lateral sinus and jugular vein. |
| Acute exacerbation, meningitis | " | Pus in tympanum. Opening through drum-head. Thrombo-phlebitis of transverse sinus. Abscess in subdural space and l. temporal lobe. Purulent lepto-meningitis of the base and convexity. |

TABULAR REPORT OF PURULENT BRAIN DEPOSITS, AND PHLEBITIS AND THROM-

| No. | Where Reported. | Surgeon's Name, etc. | Sex. | Age. | Ear. | Previous History. |
|---|---|---|---|---|---|---|
| 30 | Arch. Otology, 1880 | Frankel | M | 22 | R | Chronic otorrhœa |
| 31 | Arch. Otology, 1880 | Frankel | F | 28 | R | " " |
| 32 | Arch. Otology, 1880 | Frankel | M | 53 | L | " " |
| 33 | Arch. Otology, 1880 | Frankel | F | 3 | L | Pebble in ex. meatus; attempt at removal |
| 34 | Am. Jour. Otology, 1879 | Greene | | | | Otitis media; polypus |
| 35 | Lancet, 1878 | Gribbon | M | 22 | R | Chronic otorrhœa |
| 36 | Arch. fur Ohrenh., vol. 19 | Burkner | M | 36 | R | " " pain in ear and head; deafness |
| 37 | Arch. fur Ohrenh., vol. 19 | Burkner | M | 20 | L | Chronic bilateral otorrhœa |
| 38 | Arch. fur Ohrenh., vol. 19 | Burkner | M | 17 | L | Acute purulent otorrhœa; l. facial paralysis |
| 39 | Am. Jour. Otology, 1881 | Loring, N. Y. City | M | | R | Influenza; deafness; closure of eustachian tubes; frequent similar attacks |
| 40 | Arch. der Heilk., vol. 2 | Wendt | M | 49 | R | |
| 41 | Arch. der Heilk., vol. 2 | Wendt | M | 52 | R | Acute catarrhal otitis |
| 42 | Arch. der Heilk., vol. 2 | Wendt | | | | |
| 43 | | de Rossi, Rome | F | 57 | L | Malaria; tinnitus aurium; acute otorrhœa |
| 44 | | de Rossi, Rome | F | 18 | R | Chronic otorrhœa |
| 45 | King's Col. Hos. Reports | Pritchard, London | M | 23 | L | Chronic otorrhœa; frontal headache: pain in ear |
| 46 | King's Col. Hos. Reports | Pritchard, London | M | 26 | L | Chronic otorrhœa, pain in ear, and swelling of l. side of neck |
| 47 | Arch. Otology, 1882 | Munson, Albany | F | 39 | L | Chronic otorrhœa. Has had partial l. facial paralysis |
| 48 | Lancet, 1887 | Gray | | 26 | R | " " pain in and behind ear; facial paralysis |
| 49 | Trans. of Am. Otol. Society | Pomeroy | | | R | " " |
| 50 | Arch. of Otology, 1889 | Finlayson & Barr | M | 22 | R | " " |
| 51 | British Med. Jour., 1886 | Gowers & Barker | | 19 | R | Chronic otorrhœa; pain in and around ear |
| 52 | Arch. Otology, vol. 12 | Knapp, N. Y. City | M | 39 | R | Acute purulent otorrhœa |
| 53 | Arch. Otology, 1880 | Steinbrugge | M | 58 | R | Chronic otorrhœa |
| 54 | Breslauer Aerztl. Zeits, 1879 | Binswanger | M | 51 | R | Neither discharge from ears nor deafness has been observed |

BOSIS OF THE CEREBRAL VEINS AND SINUSES FOLLOWING EAR DISEASE.

| Present History. | Result. | Autopsy. |
|---|---|---|
| Appearance of cerebral symptoms after knock on head | Death | Pus in tympanum. No perforation of drum-head. Incus gone. Caries of petrous bone through tegmen-tympani. Abscess in r. temporal lobe. Encapsulated. Thrombo-phlebitis of r. transverse sinus. |
| R. facial paralysis, vomiting, retained urine, dilated pupils, coma | " | Pus in tympanum. Perforation of drum-head. Caries of tegmen-tympani and ex. meatus. Inspissated exudation compresses facial nerve in Fallopian canal. Purulent basilar meningitis. Abscess in r. temporal lobe. Encapsulated. Mastoid sclerosed. |
| Polypus removed; headaches; mastoid opened; meningitis | " | Epithelomia of l. middle ear, with destruction of most of temporal bone. Purulent basilar meningitis. |
| Meningitis | " | Drum-head gone. Pebble in tympanum. Pus in tympanum. Purulent meningitis of convexity. |
| Vomiting, pain, convulsions | " | Caries of tegmen-tympani. Perforation of dura-mater near transverse sinus. Abscess of temporal lobe. |
| Headache, nausea, paresis of lower extremities | " | Abscess in r. lobe of cerebellum. Caries of petrous at internal aud. meatus. Pus in mastoid. |
| Pain in ear and head; deafness; diminution of discharge; convulsions, delirium, coma; swelling over mastoid muscle | " | Necrosis in tympanum and ex. meatus. Dura near temporal bone injected, thickened, and purulent. |
| Pain in l. ear; cessation of discharge; chills, vomiting, vertigo, high fever; thrombus felt in l. jugular; pain in neck; apathy; mastoid red and swelled; facial veins enlarged | " | Thrombi in transverse sinus and bulbous venæ jugularis. Caries of jugular fossa. |
| Granulations in tympanum; removed; later had pain in ear; cessation of discharge; return of bad symptoms; contraction of l. pupil; nystagmus of both eyes; somnolence; deafness; paresis of l. leg; paralysis of l. abducens; pain in all branches of trigeminus; vomiting | " | Pus around chiasm. Anterior extremity of l. lobe of cerebellum is adherent to posterior margin of temporal bone. Abscess in l. pons. At sup. border of temporal bone are three carious openings communicating with irregular cavity involving the entire posterior portion of temporal bone. This cavity is filled with a mass which infiltrates the posterior wall of temporal bone just above sigmoid sulcus, and is also connected with vestibule. Ossicles gone. Int. ear destroyed by gelatinous mass. |
| Pain in ear and side of head; acute catarrhal otitis; delirium; drum-head punctured; no pus | Death | Congestion of dura at roof of tympanum. Sero-purulent exudation in subarachnoid space, extending from longitudinal fissure down side of brain. Pus in upper surface of r. lobe of cerebellum. Pseudo-membrane in tympanum. |
| Tinnitus aurium; pain, deafness, unconsciousness, convulsions | " | Basilar meningitis. |
| Pain; tinnitus aurium; found dead in bed | " | Diffuse meningitis. |
| Traumatic inflammation of middle ear | " | Basilar meningitis. |
| L. side of neck swelled; swelling incised and pus evacuated; pus comes from meatus on pressure of swelling; polypus in middle ear; middle ear connected by sinus with swelling; mastoid too sclerosed to open; chills | " | Coagulated blood in left sinuses. Localized meningitis. Clot in jugular vein. Caries of atlas and 2d vertebra. Pus in cavum tympanum. Occipital condyles carious. Pus in mastoid cells. Transverse sinus surrounded by pus. Carious opening between mastoid antrum and sigmoid sinus. |
| Pain in ear and side of head; fever; deafness; tympanic granulations; removed; headache, exophthalmia, ptosis; pain in head; painful swelling over mastoid muscle; coma | " | Dura adheres to bone. Pus in subdural space. Pus covers trigeminus and acoustic nerves. Dura at base covered with pus. Pus in sup. petrosal sinus, inferior cavernous sinus, and transverse sinus. Caries of tegmen-tympani. Pus in tympanum. Thrombi in longitudinal sinus and communicating veins. Abscess in inferior posterior side of frontal lobe. |
| Caught cold; increased discharge and pain; attacks of unconsciousness, with loss of speech; convulsions; twitching of l. side of face; semi-conscious; fits, drowsy, incoherent; tenderness on pressure, most marked about two inches above meatus; slight facial paralysis; operation: skull trephined two inches above and one-half inch in front of meatus; trephined again one inch behind original opening; pus found outside of dura; trephined again over occipital parietal region. No pus; dura always left intact | Recov'y | |
| Pain in ear and side of head; chills, vomiting, dizzy; aural polypus; temperature medium; delirium; twitching of l. eyebrow and angle of mouth; retention of urine; semi-conscious; operation; trephined one and one-half inches behind meatus, and the same distance above the cerebral base line; pus evacuated; improvement; delirium, chills; wound re-explored; no pus; mastoid opened; no pus; paralysis of r. arm and leg; l. optic neuritis; track of original operation freely dilated; pus escaped | Recov'y | |
| Pain in ear, vomiting, tinnitus-aurium, convulsions, right-sided | Death | Polypus of ex. meatus. Semi-circular canals carious. Abscess of middle lobe of cerebrum, directly above semi-circular canals. Opening in meninges and brain tissue connecting the semi-circular canals and abscess. |
| Fever, apathetic, half comatose, paralysis and anæsthesia of l. leg, delirium; mastoid opened; no pus, coma, hemiplegia, hemi-anæsthesia | " | Carious opening through tegmen-tympani. Abscess between dura and petrous. R. cerebral hemisphere covered with pus. Perforation of dura upon posterior surface of petrous. |
| | " | Diffused abscess of r. lobe of cerebellum. Tympanum carious. Carious opening through tegmen-tympani. |
| Acute exacerbation, fever moderate, giddy, pain in jaw and behind ear, vomiting, chills, pain in frontal and occipital regions, pain in back, head retracted, r. facial paralysis, constipation, stupor, vomiting | " | Pia congested. Purulent exudation in frontal convolution of both sides. Pus at base in region of medulla. Brain adherent near r. internal auditory meatus. Granulations in tympanum and mastoid antrum. Malleus and incus gone. Facial nerve disorganized and denuded of its bony covering. Mastoid cells obliterated and converted into sclerosed mass. Pus in antrum. Caries of tympanum. Caries of cribriform lamina. Carious aperture on upper part of petrous communicates with cochlea. Bone over sup. semi-circular canal is carious. |
| Fever, bilateral optic neuritis, vomiting, unequal pupils; mastoid opened; improvement, but unequal pupils and optic neuritis persisted; stupor, insomnia, delirium, chills; operation; skull trephined 1¾ inches behind and 1¾ inches above center of meatus; aspiration needle introduced into temporal lobe inward, forward and downward; pus evacuated | Recov'y | |
| Pain in ear and head, especially in r. occipital region; later pain, swelling and fluctuation in l. occipital region; incision at this point; pus liberated, bone denuded, wound kept open; improvement; later become worse; pus in occipital region came from interior of skull; frontal headaches, insomnia, nausea, pale, chills, fever, swelling below original opening; incised; pus found; improvement; became worse; optic neuritis both eyes; swelling appeared upward and backward from original opening; incised; pus found; probe passed into cranial cavity; pain in r. side of forehead, nausea, vomiting, delirium, coma | Death | The openings in the skull referred to in the history of the case were found. At outer surface of lateral sinus a streak of pus led along the transverse sulcus to large collection of pus at lowest part of sigmoid fossa. Pus in mastoid cells and tympanum. Abscess in middle and outer part of little brain. Not encapsulated. |
| Vertigo, pain in r. parietal region and ear, neuralgia in third branch of r. trigeminus, cholesteatomatous masses in meatus and middle ear, l. arm and leg partially paralyzed, impaired vision, constipation, coma | Death | Fluid blood in all sinuses. Pia congested. R. temp. lobe adherent to petrous. Abscess in r. temp. lobe. Encapsulated. Surrounding brain substance sclerosed. L. optic nerve atrophied. Perforation through anterior surface of r. petrous and dura. Carious opening in semi-circular canals. Drum-head and ossicles gone. Tympanum necrosed so that it and mastoid antrum are thrown into one cavity, all filled with cholesteatoma. |
| Fever, loss of appetite, vomiting, paralysis of l. arm, epilepsy, chronic convulsions, r. pupil dilated | " | Abscess in r. first frontal convolution. Encapsulated. Roof of r. tympanum inflamed. Granulations in r. tympanum and mastoid cells. Drum-head destroyed. Ossicles intact. |

TABULAR REPORT OF PURULENT BRAIN DEPOSITS, AND PHLEBITIS AND THROM-

| No. | Where Reported. | Surgeon's Name, etc. | Sex. | Age. | Ear. | Previous History. |
|---|---|---|---|---|---|---|
| 55 | Arch. fur Ohrenh., 1879, No's 11 and 12 | Kretschy | | | | |
| 56 | Arch. fur Ohrenh., 1879, No's 11 and 12 | Kretschy | | | | |
| 57 | Arch. fur Ohrenh., 1879, No's 11 and 12 | Kretschy | | | L | |
| 58 | | Burckhardt | F | 19 | L | Chronic otorrhœa |
| 59 | | Burckhardt | | 2 | L | |
| 60 | | Burckhardt | F | 7 | | |
| 61 | | Burckhardt | F | 2 | L | |
| 62 | Arch. Otology, 1879 | Hartman | F | 34 | L | Chronic otorrhœa; occasional acute exacerbations |
| 63 | Arch. Otology, 1879 | Hartman | M | 30 | L | Chronic otorrhœa; violent pain in l. half of face, which led to facial paralysis |
| 64 | Arch. Otology, 1879 | Hartman | M | 13 | R | Chronic otorrhœa |
| 65 | Lancet, 1880 | Field | M | 18 | R | " " |
| 66 | Arch. Otology, 1880 | Pooley | M | 45 | L | Blow on l. side of head, followed by deafness and otorrhœa |
| 67 | Am. Jour. Otology, 1881 | Rodman, Waterbury | M | 21 | R | Chronic otorrhœa; occasional acute exacerbations |
| 68 | Deutsche Med. Wochens., 1890 | Hoffman | | | | |
| 69 | Dublin Med. Jour., 1890 | Patterson | | | | |
| 70 | Arch. fur Ohrenh., 1879 | Burckhardt-Merian | F | 19 | L | Chronic otorrhœa |
| 71 | Arch. fur Ohrenh., 1879 | Burckhardt-Merian | | | L | " " |
| 72 | Arch. fur Ohrenh., 1879 | Burckhardt-Merian | | | | |
| 73 | Arch. fur Ohrenh., 1879 | Burckhardt-Merian | | | | Chronic otorrhœa |
| 74 | Med. Times and Gazette, 1880 | Johnson | M | 14 | R | " " |
| 75 | Med. Times and Gazette | Thompson | F | 12 | R | Tuberculous; chronic otorrhœa |
| 76 | Arch. Otology, 1888 | Ferrer, San Francisco | M | 60 | | |
| 77 | Arch. Otology, 1888 | Lewis, Birmingham | | | | |
| 78 | Arch. Otology, 1888 | Lewis, Birmingham | | | | |
| 79 | Arch. Otology, 1888 | Barr, Glasgow | M | 21 | L | Chronic otorrhœa |
| 80 | Arch. Otology, 1888 | Roosa, N. Y. City | F | 42 | L | Advanced Bright's disease |
| 81 | Brit. Med. Jour., 1888 | Braker | M | 30 | R | Chronic otorrhœa; lateral sclerosis of spinal cord |
| 82 | New York Med. Jour., 1887 | Abbe | | | | |
| 83 | Arch. fur Ohrenh., vol. 26 | Schmiegelow, Copenhagen | M | | R | Chronic catarrhal otitis |
| 84 | New York Med. Jour., 1886 | Mathewson, Brooklyn | M | 40 | R | Chronic otorrhœa |
| 85 | New York Med. Jour., 1886 | Mathewson, Brooklyn | F | 11 | L | " " tympanic polypus |
| 86 | New York Med. Jour., 1886 | Mathewson, Brooklyn | M | 5 | R | |
| 87 | British Med. Jour., 1887 | Greenfield | M | 26 | L | Deafness |
| 88 | Canada Lancet, 1881 | Ryerson | Child | | L | Scarlet fever |
| 89 | Arch. fur Ohrenh., vol. 26 | Wagenhauser | | | L | |
| 90 | Arch. Otology, 1891 | Friedenwald, Baltimore | M | 17 | L | Chronic otorrhœa; had abscess lanced behind ear years ago |

BOSIS OF THE CEREBRAL VEINS AND SINUSES FOLLOWING EAR DISEASE.

| Present History. | Result. | Autopsy. |
|---|---|---|
| . . . . . | Death . | Purulent thrombus in L. transverse sinus. Pus in jugular sinus, and defect in its anterior wall leads to necrosed petrous. Cholesteatoma in tympanum. Fibrous tissue about jugular vein infiltrated with serum and pus. |
| . . . . . | " | Purulent infiltration of inner membranes of brain. |
| . . . . . | " | Abscess in l. cerebellum. |
| Died of meningitic symptoms after illness of 16 days . . . . . | " | L. trigeminus acousticous and facialis imbedded in pus. Purulent pia-mater. Thrombus in sup. sinus. Caries of petrosal sinus canal. Polypus in tympanum. Mastoid cells absent. |
| Death from tubercular meningitis . . . . . | " | Partial necrosis of l. wall of lateral sinus. Thrombas in lateral sinus. Carious openings ot bony walls of sulcus transversus. |
| . . . . . | " | Ossicles gone. Caries of mastoid process. Caries of parietal bone. |
| . . . . . | " | Multiple caries on skull. L. drum-head gone. Malleus and incus gone. Necrosis of mastoid process. |
| Painful mastoid, fever and chills, pain in l. temple, delirium . | " | Anterior l. hemisphere covered with pus, extending to base of brain. Dura over l. tegmen-tympani discolored and pierced by openings, beneath which a cavity of pus was seen. Tegmen-tympani perforated by caries. Abscess in brain over tegmen-tympani, extending into lateral ventricle, which, with r. and middle ventricle, was filled with pus. Spinal cord bathed in pus. Polypus and necrosis in tympanum. Mastoid sclerosed. |
| Pain in entire head; polypus in l. tympanum . . . . . | Death . | Purulent meningitis at posterior cerebral fossa. Abscess in l. cerebellum. Caries of bony wall of facial canal. Caries of semi-circular canal. Mastoid sclerosed. |
| Meningitis; swollen mastoid; Wilde's incision; pus liberated; delirium, coma . . . . . | " | Pus patches over brain surface. Pus at base of brain, especially at r. of cerebellum. Abscess in middle cranial fossa. Pus at sella turcica and and l. middle cranial fossa. Necrosis of tegmen-tympani. Tympanum carious. Mastoid sclerosed. |
| Pain in head . . . . . | " | Suppurative meningitis over r. petrous bone. Inner mastoid at lateral sinus carious. Thrombus in lateral sinus. Abscess of r. tempero-sphenoidal lobe and occipital lobe. Abscess in r. cerebellum. Incus and stapes gone. |
| Painful ear; tinnitus aurium; drum-head perforated; mastoid swollen, red and painful; mastoid opened; pus found; not much fever . . . . . | " | Abscess in l. middle lobe. L. sided pachymeningitis. Petrous carious, especially at outer half of middle surface. Pus in lateral ventricle of r. side of brain. |
| Mastoid symptoms; mastoid opened; pus found . . . . . | | Meningitis. Pus in neck between mastoid and styloid process. Caries in inner plate of mastoid at sulcus of lateral sinus. Caries of squamous. Lower surface of mastoid has 3 carious perforations in the groove next the tip of mastoid in the digastric fossa, all communicating with the carious mastoid cells. Mastoid antrum carious. |
| He discovered in the course of an operation for mastoid abscess, an abscess in the occipital lobe, which he opened . . . . . | Recov'y | |
| Pyæmia, following a suppurating ear . . . . . | Death . | Thrombus in lateral sinus. |
| Meningitis . . . . . | " | L. circumscribed basilar meningitis. Fistula between mastoid antrum and sup. petrosal sinus. Polypus in tympanum. No mastoid cells. |
| . . . . . | " | Caries of tympanum, antrum, mastoid cells, and osseous walls of lateral sinus. Thrombosis of lateral sinus. Tubercular meningitis. |
| . . . . . | " | Caries of tympanum and antrum. Carious opening through outer wall of mastoid. Caries of parietal bone. Meningitis. |
| . . . . . | " | Necrosis of antrum and squamous. Meningitis. |
| Chills, headache, thoracic pains, unsteady gait, constipation, night sweats, pupils dilated, delirium, coma, temp. and pulse not high . . . . . | " | Double pleurisy. Pus in lungs, r. internal jugular vein, and tympanum. Abscess at apex of petrous bone. Necrosis of petrous near labyrinth. Blood coagulum in sigmoid sinus. |
| Headache, vertigo, vomiting, dilated pupils, temp. and pulse medium, chills, involuntary defecation, cough, thoracic pains, coma, convulsions . . . . . | " | Congestion of pia-mater of r. cerebellum adherent to dura. Abscess in r. cerebellum. Tubercular deposits in brain. Thrombus in r. internal jugular vein, sub-clavian vein, vena-cava, and r. auricle. Pus in r. half of posterior occipital fossa, r. sigmoid sinus, tympanum, vestibule, and semi-circular canals. Lungs abscessed. |
| Mastoid abscess; opened mastoid; found pus; death from hematemesis . . . . . | " | Pus and granulations in mastoid cells and tympanum. Meningitis. |
| Mastoid abscess . . . . . | " | Abscess in temporo-sphenoidal lobe. |
| Mastoid abscess . . . . . | " | Phlebitis of lateral sinus. |
| Headache, slow and intermittent pulse, normal or sub-normal temp., contraction of l. pupil, paresis of all ocular muscles except external rectus, partial r. facial paralysis, paresis of r. arm with wrist-drop; mastoid opened; no pus; skull trephined above ex. meatus; brain pierced with trocar; pus found . . . . . | Recov'y | |
| Acute otitis; drum-head lanced; pain and tenderness at junction of temporal and occipital bones; temp. medium . . . . . | Death . | Caries of tympanum. Pus in middle ear. 7th nerve exposed and imbedded in pus. No mastoid disease. Meningitis. |
| Acute exacerbation, pain in r. half of head, vomiting, epilepsy, contraction of r. pupil; mastoid opened; no pus, emaciation, incontinence of urine, stupor, somnolence; operation; parietal bone trephined; duru incised; no pus; trephined again, 1½ inches above and 1¼ inches behind the ex. meatus; needle passed 1½ inches deep, forward and inward; pus found . . . . | Recov'y | |
| Acute otitis media; acute meningitis; choked disc on same side . . . . . | Death . | Purulent meningitis at base of cerebrum and lower surface of cerebellum. No evidence that pus had extended from ear. Internal auditory canal showed streaks of pus following the course of nerve. |
| Neuralgia of all three branches of r. trigeminous, followed by r. facial paralysis; r. choked disc; later, purulent otitis, pain, mastoid inflamed; mastoid opened; pus found; œdema of r. side of face; skull trephined at posterior cranial fossa; no pus; mastoid opening enlarged backward; improvement, nausea, fever, headache, vomiting, unconsciousness . . . . . | " | Diffuse purulent lepto-meningitis. Abscess in temp. lobe. |
| Pain over mastoid; Wilde's incision; brain complications . . . | " | Caries of tegmen-tympani. Abscess of brain over tympanum. |
| Spontaneous opening in ex. mastoid plate; facial paralysis; improvement; became worse; pain, vomiting, drowsiness, constipation, optic neuritis, convulsions . . . . . | " | Dura-mater adherent to petrous. Pus in cerebellum. |
| Acute otitis media; drum-head not perforated . . . . . | " | Caries of tegmen-tympani. Basilar meningitis. |
| Headache, vomiting, ptosis, optic neuritis, sub-normal temp., semi-comatose; operation; skull trephined; dura incised; pus found . . . . . | Recov'y | |
| Acute purulent otorrhœa; mastoid pain, swelling and tenderness; pain in head; ptosis r. eye; divergent squint right eye; both pupils dilated; coma; mastoid opened; pus found; improvement; later became worse; vision poor; optic discs swollen; fever, delirium . . . . . | Death . | Dura adherent to skull. Pus on surface of both sup. lobes. Purulent thrombi in lateral sinus. |
| Acute otorrhœa; mastoid operation . . . . . | " | Purulent meningitis. Inner plate of mastoid carious and perforated. Inner surface of petrous covered with granulations. |
| Pain, dizziness, aural polypi, left-sided deafness, chills, headache, temp. and pulse medium, tenderness behind ear; later, chills and high fever; operation after Von Bergmann, viz.: from a point 4 c.m. behind ex. meatus in a line made with the lower margin of the orbit, ascend perpendicularly for 5 c.m. to reach point for trephening; no pus; mastoid opened; pus and cholesteatoma found . . . . . | " | Pus in pia, covering surface of l. frontal and parietal lobes. Perforation of inner mastoid plate. Purulent thrombus in lateral sinus. |

TABULAR REPORT OF PURULENT BRAIN DEPOSITS, AND PHLEBITIS AND THROM-

| No. | Where Reported. | Surgeon's Name, etc. | Sex. | Age. | Ear. | Previous History. |
|---|---|---|---|---|---|---|
| 91 | Arch. Otology, 1879 | Pierce | F | 34 | L | Chronic otorrhœa |
| 92 | Trans. Am. Otolo. Society | Sexton, N. Y. City | | | | |
| 93 | Am. Otolo. Society, 1887 | Roosa, N. Y. City | M | 11 | R | |
| 94 | Med. Times, 1885 | Parker | | | R | |
| 95 | Arch. Otology, 1884 | Sutphen | M | 44 | B | Chronic otorrhœa |
| 96 | Arch. Otology, 1884 | Sutphen | M | 21 | R | " " |
| 97 | Arch. Otology, 1881 | Moss, Heidelberg | M | 23 | L | " " polypus; paralysis l. facial nerve; deafness l. ear |
| 98 | Arch. Otology, 1884 | Hedinger | M | | L | |
| 99 | Arch. Otology, 1884 | Hedinger | F | | L | |
| 100 | Arch. Otology, 1885 | Rothholz | M | 20 | R | Chronic otorrhœa |
| 101 | Arch. Otology, 1886 | Sutphen | M | 25 | R | Chronic otorrhœa; from blow on ear; four years before had abscess behind ear, which was opened and healed; swelling recently recurred, with pain |
| 102 | Arch. Otology, 1880 | Pooley | M | 30 | R | Chronic otorrhœa |
| 103 | Arch. Otology, 1887 | Barr & McEwen, Glasgow | M | 9 | R | " " |
| 104 | Arch. Otology, 1889 | McEwen, Glasgow | M | 17 | L | Chronic otorrhœa |
| 105 | British Med. Jour., 1879 | Barr, Glasgow | M | 17 | L | " " |
| 106 | Glasgow Med. Jour., 1880 | Barr, Glasgow | M | 14 | L | " " |
| 107 | Glasgow Med. Jour., 1880 | Barr, Glasgow | " | 17 | L | " " |
| 108 | Glasgow Med. Jour., 1880 | Barr, Glasgow | " | 12 | L | " " |
| 109 | | Remmel | | | R | " " |
| 110 | | Taylor | | | | " " |
| 111 | | Moss, Heidelberg | | | R | |
| 112 | | Moss, Heidelberg | | | R | Chronic otorrhœa |
| 113 | | Moss, Heidelberg | | | R | " " |
| 114 | | Moss, Heidelberg | | | | " " |
| 115 | | Burckhardt-Merian | | 1 | L | " " |
| 116 | Am. Otolo. Society | Kipp | F | 28 | L | |
| 117 | Med. Corr. Blatt. von Wurttemburg, 1889 | Koebel, Stuttgart | | | | Chronic otorrhœa |

BOSIS OF THE CEREBRAL VEINS AND SINUSES FOLLOWING EAR DISEASE.

| Present History. | Result. | Autopsy. |
|---|---|---|
| Hard swelling in front of ear; granulations in meatus concealed; necrosis; pain in l. cheek and parietal bone; l. facial paralysis; pain in vertex and occipital region; carious bone discharged from ear; meatus very carious; later, the entire area of disease had fallen in, comprising the ex. meatus up to the level of the drum-head, exposing the tempero-maxillary articulation; condyle of the ramus of the jaw now exposed. The area of disease was now almost large enough to admit the closed fist; hernia cerebri appeared; paralysis of r. side with aphasia; constipation | Death . | Destruction of bony and other tissues terrific; even aqueductus fallopii, semi-circular canal and carotid canal were exposed. Hernia of tempero-sphenoidal lobe. |
| Purulent otorrhœa; caries of attic, antrum and tympanum; lymphadenoma; facial paralysis | Death . | Inner wall of attic gone. Semi-circular canals exposed. Caries of antrum and mastoid cells. Purulent meningitis. |
| Painful swelling over ear; abscess opened; no fistula | " | Abscess of tempero-sphenoidal lobe. Encapsulated. Drum-head perforated. Upper meatus necrotic. Necrosis at juncture of mastoid and squamous. Caseous pus in meatus and mastoid cells. |
| Abscess above ear; opened; convulsions, coma. | " | Necrosis of floor of middle temporal fossa, corresponding to position of middle ear. Adhesion between brain and dura. Perforation of dura. Abscess in temp. lobe. Mastoid cells filled with cheesy debris. |
| Acute exacerbation, pupils contracted, aphasia, 7th nerve on l. side paralyzed, choked discs both eyes, necrosis l. ex. meatus, paralysis r. arm and leg; opening made into cranial cavity by way of meatus and mastoid; no pus found | " | Abscess in anterior and middle lobes of l. hemisphere. Encapsulated. Necrosis of petrous. |
| Acute exacerbation, paralysis of r. abducens, impaired vision of both eyes, both discs swollen, fever, chills, coma; probe can be passed into cranial cavity by way of upper meatus; no pus. | " | Thrombi in r. lateral and sup. longitudinal sinuses. Caries in sulcus for lateral sinus. Necrosis of sup. wall of petrous. Caries of meatus, tympanum, and walls of mastoid. |
| Vertigo, coma | " | Abscess in l. cerebellum. Encapsulated. |
| Acute purulent otorrhœa, fever, fluctuating swelling of l. parotid gland; opened; found pus; pus may be forced from swelling into ex. meatus; coma | " | Pus under dura and arachnoid, and in antrum and tympanum. Purulent thrombus in sup. petrosal sinus. Ulcer in this sinus communicating with pyramid. Carious opening connects cranial cavity and tympanum, and mastoid antrum. Ulcer in transverse sinus. Ex. meatus carious. |
| Acute purulent otorrhœa, fever, polypus; removed; granulations; swelling of glands below ear, and frequent attacks of pain in entire l. half of head; vertigo; death from tuberculosis | " | Ex. meatus carious. Between bulbous venæ jugularis and bony portions of Eustachian tube there were two fistulas leading into pyramid. Tympanum carious and purulent. Internal wall of carotid canal carious. Thrombus in carotid artery. Cheesy pus in mastoid antrum. |
| Pain in vertex, deaf, drum-head gone, polypus, partial paralysis of auditory nerve, temp. and pulse nearly normal, constipation, stupor, delirium, unconsciousness, divergent strabismus, herpes on r. cheek | " | Dura congested. Purulent lepto-meningitis, especially at base. Communicating abscess in r. cerebellum. Encapsulated. Drum-head gone. Pus and polypus in tympanum. Ossicles gone. Chorda-tympani destroyed. Pus in Fallopian canal. Facial nerve exposed in tympanum. Facial nerve looks normal in tympanum, but from gangliform swelling to internal auditory meatus it is inflamed. Acoustic nerve presents the same general appearances. Pus in vestibule and cochlea. Petrous bone saturated with pus. |
| Meatus swelled; granulations and necrosis in tympanum; abscess opened; communication with tympanum established; improvement; later became worse; a fistulous opening existed just below ex. meatus, through which pus escaped; carious bone in this fistula; water injected into meatus escaped into mouth and fistula; vomiting, headache, aural hæmorrhage, fever, pain, delirium | Death . | The bone in region of middle ear has been changed into one large carious cavity. Opening into internal carotid artery. Cerebellum abscess communicating with carious cavity. |
| Pain in r. ear and r. side of head; mastoid swollen and tender; deaf; Wilde's incision; carious bone found; trephining refused; chills, delirium; mastoid opened; pus found; coma | Death . | Meningitis at convexity and base. Red inflammatory softening at apex of temporal bone. Upper surface of cerebellum inflamed. Pus in pia-mater, arachnoid, tympanum, mastoid antrum and cells, vestibule, semi-circular canals, Eustachian tube, and canal for tensor-tympani. Necrosis in tympanum. Incus and stapes gone. |
| Acute exacerbation; mastoid abscess; opened; found pus; no improvement; fever slight; ptosis of r. eye; paresis of l. internal rectus and l. orbicularis; veins r. side of head congested; r. mastoid muscle rigid; pain on pressure of vein passing through posterior condyloid foramen; dense stupor; pulse slow and feeble; constipation; operation; skull trephined 1½ inches above and ½ inch behind the center of ex. meatus; dura opened; dura and pia congested; aspiration needle passed toward eminence of petrous bone; pus found ¾ inch deep; at same time skull again trephined just above osseous boundary of ex. meatus, involving squamo-petrosal suture; abscess reached | Recov'y | |
| Unconscious, weak and slow pulse, optic neuritis, nearly moribund, carious sinus into mastoid cells, vomiting, pain in head, chills, l. hemiplegia; mastoid opened, carious matter expelled, lateral sinus exposed, on which were found granulations; the bone was then perforated further back than the groove for lateral sinus; pus escaped from over the cerebellum | Recov'y | |
| Vomiting, pain in head, stupor, tremors, convulsions | Death . | L. tempero-sphenoidal lobe adherent to bone beneath. Abscess in tempero-sphenoidal lobe. Encapsulated. Two carious openings in temporal bone; one through tegmen-tympani, the other in groove for lateral sinus, communicating with mastoid cells. |
| Vomiting, pain, coma, spasmodic contraction of flexors of arms and legs | " | Abscess in temporal lobe. Polypus in tympanum. Drum-head and stapes gone. |
| Aphasia, constipation, unconsciousness, paresis of r. side, coma | " | Abscess in l. temporal lobe. Carious fistulæ in roof of antrum, sigmoid flexure, and posterior wall of exterior meatus. All fistulæ communicate with mastoid cells. |
| Pain in mastoid and occiput, chills, vomiting, constipation | " | Pus beneath dura on posterior surface of l. petrous bone. The walls of the l. lateral sinus were thickened and detached from the bone by underlying pus. Mastoid cells filled with cheesy pus. |
| Temp. and pulse medium, pain in head and neck, œdema and tenderness over mastoid, r. facial paralysis, œdema of r. upper eyelid, delirium, anæsthesia r. half of face, diarrhœa | " | Caries of tympanum. Phlebitis of jugular vein. Thrombus in r. lateral sinus, extending in through inferior petrosal sinus to r. cavernous sinus, thence through circular sinus to l. cavernous sinus, which was filled with disorganized clots and pus. |
| Delirium, strabismus, diplopia, sudden rise and fall of temp., retinal veins large and tortuous | " | Thrombosis of lateral sinus and jugular vein. Lung abscess. |
| Acute otitis, pain in r. forehead and temple, chills, constipation, somnolence, impaired vision, delirium, sudden rise and fall of temp | " | Thrombosis r. lateral sinus and internal jugular vein. Phlebitis of emissary mastoid vein. |
| Pain in r. side of head | " | Thrombosis of sigmoid flexure and r. lateral sinus. Lepto-meningitis. |
| Chills, pain in occipit, vomiting | " | Phlebitis and thrombosis of lateral sinus. Meningitis. Cholesteatoma in mastoid cells. |
| Pain in ear, forehead and occiput, fever, œdema of temple, headache, stupor, spasms of upper extremities | " | Phlebitis and thrombosis of lateral and sup. petrosal sinuses. Circumscribed basilar meningitis. |
| External opening made over mastoid, vomiting, delirium, nose-bleed | " | Tubercular meningitis. Thrombosis of l. lateral sinus. |
| Furuncle l. meatus, pain in head | " | Inflammation of arachnoid and pia-mater. Abscess anterior portion of cerebellum. L. auditory and facial nerves imbedded in pus. Pus in mastoid cells. Slight tympanic inflammation. |
| Pain, facial paralysis; mastoid opened; caries of cells, antrum and tympanum; pyæmia | " | Carious openings into middle cerebral fossa and into transverse sinus. Phlebitis and thrombus of transverse sinus. |

TABULAR REPORT OF PURULENT BRAIN DEPOSITS, AND PHLEBITIS AND THROM-

| No. | Where Reported. | Surgeon's Name, etc. | Sex. | Age. | Ear. | Previous History. |
|---|---|---|---|---|---|---|
| 118 | Medical Record, 1877 | Roosa, N. Y. | M | 41 | R | |
| 119 | Am. Otolo. Society, 1871 | Greene | " | 25 | . | Chronic otorrhœa |
| 120 | Roosa on the Ear, p. 532 | Roosa, N. Y. City | . | . | L | " " mastoid abscess when young |
| 121 | Roosa on the Ear, p. 532 | Roosa, N. Y. City | . | . | R | |
| 122 | Treat. on Ear, by Roosa | Cooper | M | 65 | " | |
| 123 | Am. Otolo. Society | Roosa, N. Y. City | " | 25 | " | Chronic otorrhœa |
| 124 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 66 | " | " " deafness on one side |
| 125 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 30 | . | " " |
| 126 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 22 | . | " " |
| 127 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | F | 41 | R | " " |
| 128 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | M | 23 | " | " " |
| 129 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 25 | " | " " |
| 130 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | F | 23 | " | " " |
| 131 | Treat. on Ear | Rep. by Gull & Sutton | " | 20 | " | " " |
| 132 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | M | 13 | R | " " |
| 133 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 28 | L | " " |
| 134 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 27 | . | " " |
| 135 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 8 | L | " " |
| 136 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | F | 26 | R | " " |
| 137 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 51 | L | " " |
| 138 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 23 | " | Epilepsy |
| 139 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | M | 54 | R | " |
| 140 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | F | 7 | L | Chronic otorrhœa; great debility |
| 141 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | M | . | R | " " |
| 142 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | F | 26 | " | " " |
| 143 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 9 | L | " " |
| 144 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | M | 32 | . | " " |
| 145 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 35 | R | " " caries of mastoid; polypus in ex. meatus |
| 146 | Treat. on Ear, by Roosa | Rep. by Gull & Sutton | " | 13 | " | " " |
| 147 | Treat. on Ear, by Roosa | Greene | " | 28 | " | " " paralysis of muscles of face after walk in rain. |
| 148 | Treat. on Ear, by Roosa | Greene | " | 22 | " | Acute purulent otorrhœa |
| 149 | Treat. on Ear, by Roosa | Farwick | F | 36 | L | Chronic otorrhœa |
| 150 | Treat. on Ear, by Roosa | Schwartze | " | 18 | " | " " pain in ear |
| 151 | Treat. on Ear, by Roosa | Schwartze | M | 3 | " | " " |
| 152 | Treat. on Ear, by Roosa | Schwartze | F | 54 | R | " " |
| 153 | Treat. on Ear, by Roosa | Von Troltsch | M | 53 | L | " " polypus |
| 154 | Treat. on Ear, by Roosa | Von Troltsch | " | 24 | " | " " |
| 155 | Treat. on Ear, by Roosa | Von Troltsch | F | 21 | R | " " |
| 156 | Treat. on Ear, by Roosa | | " | 20 | L | " " |
| 157 | Treat. on Ear, by Roosa | Cock | M | 35 | . | " " |
| 158 | Treat. on Ear, by Roosa | Cock | . | 20 | . | " " |
| 159 | Treat. on Ear, by Roosa | Schwartze | F | 4 | R | " " |
| 160 | Treat. on Ear, by Roosa | Schwartze | " | 34 | " | " " |
| 161 | Treat. on Ear, by Roosa | Schwartze | . | 1 | L | " " |
| 162 | Ophthalmic Record, March, 1892 | J. Morrison Ray, Louisville, Ky. | F | 47 | R | Acute purulent otorrhœa |
| 163 | Politzer on the Ear, page 538 | R. Chimani | M | 30 | " | Chronic otorrhœa |
| 164 | Politzer on the Ear, page 539 | Burckhardt-Merian, Basle | F | 25 | L | Chronic otorrhœa |
| 165 | | J. Orne Greene | " | . | R | Chronic otorrhœa |
| 166 | Edinburgh Med. Jour., Nov., 1879 | Kirk Duncanson | M | 37 | L | Acute otorrhœa |
| 167 | Arch. Otol., April, 1892 | C. Truckenbrod, Hamburg | " | 54 | " | Deafness and tinnitus for ten days |

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BOSIS OF THE CEREBRAL VEINS AND SINUSES FOLLOWING EAR DISEASE.

| Present History. | Result. | Autopsy. |
|---|---|---|
| Acute purulent otorrhœa; meningitic symptoms, chills, temp medium, pulse low; Wilde's incision; delirium, pyæmia | Death | Fibrino-purulent exudation in pia. Coagula in sinuses. Lateral ventricle dilated and filled with blood-stained serum. Tegmen-tympani carious. Lateral sinus carious. |
| Deaf, dizziness, headache, diplopia, partial paralysis, coma | " | Abscess in tempero-sphenoidal lobe. Necrosis tegmen-tympani. Sinus connecting auditory canal and cerebral cavity. |
| Pain, high fever | " | Pus under dura and in mastoid cells. Temporal bone infiltrated with pus. Drum-head, malleus, incus, and part of stapes gone. |
| Acute purulent otorrhœa, pain in r. side of head, delirium, retention of urine, temp. and pulse medium | " | R. optic nerve atrophied. Meninges at base of cerebellum and upper part of spinal cord, covered with lymph and sero-pus. Malleus and incus gone. |
| Acute purulent otorrhœa, pain behind ear and in head, stupor. | " | Dura congested. Lymph at base. Pus at base, extending to medulla. |
| Pain in head and ear, temp. and pulse medium, chills, pleurisy, pneumonia, pain over lateral sinus, exophthalmus | " | Thrombus of r. internal jugular. Pus in r. lateral sinus. Drum-head and ossicles gone. R. lateral sinus carious. |
| Paralysis of one side, ptosis, giddy, chills, drowsy, delirium, face flushed, head hot, convulsions | " | Abscess in center of r. cerebral hemisphere. |
| Head and neck rigidly curved forward, and spine curved; rotary movements of head; unable to swallow | " | Abscess in pons varolli. |
| Sore throat for one week, and became generally ill; chills, semi-coma | " | Abscess in middle lobe. |
| Paralysis of r. half of face; pain in r. side of head, drowsy, semi-coma | " | Abscess in middle half of r. lobe of cerebellum, communicating directly with diseased temporal bone. |
| Caries of temporal bone, chills, pain in head, vomiting | " | Abscess in r. lobe of cerebellum. |
| Frontal headache, vertigo, delirium, paresis of l. side, coma | " | Abscess in r. middle lobe of cerebrum. |
| Pain in r. side of head and r. ear, vomiting | " | Suppuration and sloughing of r. middle lobe of hemisphere. |
| Paralysis of r. 7th nerve, pain in head, pain on moving neck, chills, nausea, vomiting, sweating | " | Abscess in cerebellum. |
| Syncope, convulsions with insensibility, pain in head, nausea, delirium, convulsions, pain and cramp in l. leg, coma | " | Abscess of the posterior lobe of r. hemisphere. |
| Delirium, coma | " | Abscess in anterior and middle lobe of l. hemisphere, communicating with carious petrous. |
| Pain in head, paralysis of r. side of face; death from hæmorrhage from lateral sinus | " | Dura inflamed. Sloughing of brain tissues. Lateral sinus inflamed and sloughy. |
| Vomiting, convulsions, paralysis of l. upper eyelid, limbs weak, pain in ear, dull, drowsy, semi-coma, convulsions | " | Abscess in l. cerebral hemisphere. |
| Delirium, opisthotonos, coma | " | Abscess in under surface of middle cerebral lobe. |
| Cough, pain in limbs, pulse quick, convulsions, coma | " | Abscess in l. cerebral hemisphere. |
| Convulsions, pain in head, fever, agony, convulsions | " | Abscess. Coagulum of fibrin and blood in l. lateral sinus. |
| Pain in forehead, stupor, lost consciousness and sensibility, convulsions | " | Abscess in middle lobe of r. hemisphere. |
| Debility, epilepsy | " | Abscess in under part of l. lobe of cerebellum. |
| Cessation of discharge, chills and collapse, pain in r. side, stupid, coma | " | Abscess in r. middle lobe. |
| Pain in ear, headache, dizzy, coma. | " | Abscess in upper part of r. cerebral hemisphere. |
| Fever, vomiting, pain in ear, l. sided paralysis, coma | " | Abscess in middle lobe of l. side of cerebrum. |
| Chills, fever, abscess behind ear, stupor, convulsions | " | Abscess in middle cerebral lobe. Pus between diseased mastoid and dura-mater. |
| Pain in back of head, neck and shoulders of r. side, stupor, coma | " | Abscess in r. lobe of cerebellum. |
| Fever, headache, thick speech, hemiplegia, vomiting, drowsy, pain, stupor | " | Three abscesses in r. lobe of cerebellum. |
| Pain in r. side of head, vertigo, chills, nausea, vomiting, coma. | " | Abscess in r. half of cerebellum. Roof of tympanum bare, but not carious. |
| Pain in ear, headache, difficulty in swallowing, vertigo, paralysis of r. hypoglossal nerve | " | Meningitis. Caries of inner table of skull. Cochlea and semi-circular canals filled with solid red mass. |
| Pain in ear and l. side of head, vertigo, delirium | " | Abscess in l. cerebral hemisphere. Caries of roof of tympanum. |
| Chills, left side of neck œdematous and painful on pressure, especially over jugular; painful swallowing, nausea, uvula œdematous, vomiting, singultus, convulsion of l. arm | " | Clots in sup. longitudinal and sup. petrosal sinus. Old thrombus in l. lateral sinus. |
| Meningitis | " | Œdema of pia-mater. L. lateral sinus contained a thrombus. Carious bone in l. auditory canal. |
| Pain in ear and head, vomiting, vertigo, coma, ptosis of r. eye | " | Hyperæmia of membranes of brain. Œdema of pia-mater. Thrombus in r. sup. petrosal sinus. |
| Pain in ear and head, coma | " | Abscess in l. middle cerebral lobe, connecting with petrous bone. |
| Brain symptoms | " | Carious perforation of roof of tympanum. Abscess of l. inferior cerebral lobe. |
| Chills, œdema near ear, swelling of sub-maxillary glands, delirium, r. pupil dilated, coma | " | Thrombus in r. lateral sinus. Caries of roof of tympanum. |
| Headache, vomiting, pain in ear and occiput, coma, delirium | " | Abscess in l. cerebellum and in l. inferior cerebral lobe. |
| Pain in ear | " | Sinuses congested. R. lateral sinus filled with a clot. |
| Mastoid fistula, paralysis of facial nerve, brain symptoms | " | Caries. Phlebitis. Lateral sinus filled with pus. Gangrene of brain. |
| Chills, vomiting, pain in r. side of head, facial and pharyngeal paralysis | " | Meningitis basilaris. Abscess in r. middle lobe of cerebrum. Caries. |
| Mastoid fistula, vomiting, inanition, pneumonia, convulsions | " | Fistula of mastoid. Abscess in r. middle lobe of cerebrum. Tegmen-tympani discolored and soft. Caries of mastoid cells. |
| Temp. and pulse medium, pain in r. side of head, delirium, divergent squint, dilated pupils, coma | " | Exostosis of petrous bone. Caries of tympanum and mastoid. Connective tissue growths in vestibule. |
| Pain in mastoid and occiput, vertigo, high fever, later tumor formed over mastoid, extending to center of parietal and occipital bones; incision; pus found, underlying bones roughened; later, boring pains and heat flashes, disturbed vision, exophthalmus; later, pneumonia, jaundice, vomiting, delirium, unconsciousness, collapse | " | Purulent meningitis of frontal lobes. Pia opaque. Sulci filled with pus. Pus in tympanum. |
| Pain in l. side head and mastoid, Wilde's incision, no pus; later, collapse, swelling of l. eye-lid, exophthalmus, pupil sluggish; mastoid opened, found pus; later, swelling of r. eye-lid and r. exophthalmus; upper eye-lid incised, found pus | " | Caries of r. petrous. Rupture of sigmoid sinus. Thrombus of r. transverse sinus, both carotid sinuses, circular sinus of Ridley, l. cavernous sinus, sup. petrosal sinus, and r. ophthalmic vein. Purulent degeneration of thrombi, and inflammation of walls of above named sinuses. Purulent infiltration of connective tissue of r. orbit. Chronic œdema of pia-mater and arachnoid. Numerous pneumonic and gangrenous centres in both lungs. |
| Pain in ear, vomiting, dizziness, chills, meatus swollen, pain and œdema over mastoid, and especially over mastoid foramen and adjacent occipital bone, temp. and pulse elevated; operation; mastoid opened, no pus, improved; later, pain and swelling over mastoid muscle | " | Cholesteatoma of tympanum, mastoid cells, transverse sinus, and incisura mastoidea. Thrombo-phlebitis of l. transverse sinus and jugular veins. Basilar meningitis. Ichorous coagulum in inferior petrosal sinus and cavernous sinus. Phlebitis of ophthalmic veins on both sides. |
| Swelling and redness over mastoid; pus came from an opening one-quarter of an inch in front of the membrana tympani; pressure over mastoid caused the pus to flow more freely; operation refused | " | Purulent swelling over clavicle. Purulent mastoid periostitis. Sulcus of lateral sinus carious. Carious opening in tegmen-tympani. Thrombus in lateral sinus. Pus under dura-mater at sinus. |
| Diagnosis: Exudation in tympanum; made a paracentesis; removed serum later; a furuncle developed in the meatus; tympanic discharge became purulent; later, caught cold; pain in ear and head; temp. 104°; enlarged opening in drum-head; later, opening again enlarged; continued pain in head; vertigo, paresis of r. facial; aphasia; temp. medium; paresis of r. arm; operation; mastoid opened; no pus, no necrosis; roof of mastoid cavity opened, dura exposed; no pus; dura opened and brain exposed; no pus; exploratory puncture made, forward and upward, and pus evacuated; orifice enlarged by knife, and drainage tube inserted; cavity antiseptically irrigated; iodoform gauze used | Recov'y | Pus in arachnoid space all over brain. Dura healthy. Upper anterior surface of petrous carious. |

TABULAR REPORT OF PURULENT BRAIN DEPOSITS, AND PHLEBITIS AND THROM-

| No. | Where Reported. | Surgeon's Name, etc. | Sex. | Age. | Etc. | Previous History. |
|---|---|---|---|---|---|---|
| 168 | Arch. Otol., April, 1892 . . . . . | C. Truckenbrod, Hamburg . | M | 29 | L | . . . . . |
| 169 | Arch. Otol., April, 1892 . . . . . | C. Truckenbrod, Hamburg . | M | 25 | L | . . . . . |

*Case 154.*—Extract from Treatise on the Ear, by Roosa. Treated by Von Tröltsch. Male, age twenty-four. Left ear. Chronic otorrhœa; brain symptoms. Death.

*Autopsy.*—Carious perforation of roof of the tympanum. Abscess of the left inferior cerebral lobe.

*Case 155.*—Extract from Treatise on the Ear, by Roosa. Treated by Von Troltsch. Female, age twenty-one. Right ear. Chronic otorrhœa; chills; œdema in the vicinity of the ear; swelling of submaxillary glands; delirium; dilatation of right pupil; coma. Death.

*Autopsy.*—Thrombus in right lateral sinus. Caries of roof of tympanum.

*Case 156.*—Extract from Treatise on the Ear, by Roosa. Surgeon's name omitted. Female, age twenty. Left ear. Chronic otorrhœa; headaches; vomiting; pain in ear and occiput; coma; delirium. Death.

*Autopsy.*—Abscess in left cerebellum and in left inferior cerebral lobe.

*Case 157.*—Extract from Treatise on the Ear, by Roosa. Treated by Cock. Male, age thirty-five. Chronic otorrhœa; pain in ear. Death.

*Autopsy.*—Sinuses congested. Right lateral sinus filled with a clot.

*Case 158.*—Extract from Treatise on the Ear, by Roosa. Treated by Cock. Sex not given. Age twenty. Chronic otorrhœa; symptoms not given.

*Autopsy.*—Caries. Phlebitis. Lateral sinus filled with pus. Gangrene of the brain.

*Case 159.*—Extract from Treatise on the Ear, by Roosa. Treated by Schwartze. Female, age four. Right ear. Chronic otorrhœa; fistula of mastoid; paralysis of facial nerve; brain symptoms. Death.

*Autopsy.*—Meningitis basilaris. Abscess in right middle lobe of cerebrum. Caries.

*Case 160.*—Extract from Treatise on the Ear, by Roosa. Treated by Schwartze. Female, age thirty-four. Right ear. Purulent otorrhœa; chills; vomiting; pain in right side of head; facial and pharyngeal paralysis. Death.

*Autopsy.*—Fistula of mastoid. Abscess in right middle lobe of cerebrum. Tegmen-tympani discolored and soft. Caries of mastoid cells.

*Case 161.*—Extract from Treatise on the Ear, by Roosa. Treated by Schwartze. Sex not given, age eight months. Left ear. Purulent otorrhœa; fistula of mastoid; vomiting; inanition; pneumonia; convulsions. Death.

*Autopsy.*—Exostosis of petrous bone. Caries of tympanum and mastoid. Connective tissue growths in vestibule.

*Case 162.*—*Ophthalmic Record*, March, 1892. Treated by J. Morrison Ray, of Louisville, Ky. Female, age 47. Right ear. Acute purulent otorrhœa; temperature and pulse medium; pain in right side of head; delirium; ptosis; divergent squint; dilated pupils; coma. Death.

*Autopsy.*—Purulent meningitis of frontal lobe. Pia opaque. Sulci filled with pus. Pus in tympanum.

*Case 163.*—Politzer on the Ear, page 538. Treated by R. Chimani. Male, age 30. Right ear. Chronic otorrhœa; pain in mastoid and occiput; vertigo; high fever. Later, tumor formed over mastoid, extending to center of parietal and occipital bones; incision; pus found; underlying bones roughened. Later, boring pains and heat flashes; disturbed vision; right exophthalmus. Later, pneumonia; jaundice; vomiting; delirium; unconsciousness; collapse. Death.

*Autopsy.*—Caries of right petrous. Rupture of sigmoid sinus. Thrombus of right transverse sinus, both carotid sinuses, circular sinus of Ridley, left cavernous sinus, superior petrosal sinus, and right ophthalmic vein. Purulent degeneration of the thrombi, and inflammation of the walls of the above named sinuses. Purulent infiltration of the connective tissue of the right orbit. Chronic œdema of pia-mater and arachnoid. Numerous pneumonic and gangrenous centers in both lungs.

*Case 164.*—Politzer on the Ear, page 539. Treated by Burkhardt-Merian, of Basle. Female, age twenty-five. Left ear. Chronic otorrhœa; pain in left side of head, and mastoid; Wilde's incision; no pus. Later, collapse; swelling of left eye-lid; exophthalmus; pupil sluggish; mastoid opened; pus found. Later, swelling of right eye-lid, and right exophthalmus; upper eye-lid incised; found pus. Death.

*Autopsy.*—Cholesteatoma of tympanum, mastoid cells, transverse sinus, and incisura mastoidea. Thrombo-phlebitis of left transverse sinus and jugular veins. Basilar meningitis. Ichorous coagulum in inferior petrosal sinus and cavernous sinus. Phlebitis of ophthalmic veins on both sides.

*Case 165.*—Treated by J. Orne Greene. Female. Right ear. Chronic otorrhœa; pain in ear; vomiting; dizziness; chills; meatus swollen; pain and œdema over mastoid, and especially over mastoid foramen and adjacent occipital bone. Temperature and pulse elevated.

*Operation.*—Mastoid opened; no pus; improved. Later, pain and swelling over mastoid muscle. Death.

*Autopsy.*—Purulent swelling over clavicle. Purulent mastoid periostitis. Sulcus of lateral sinus carious. Carious opening in tegmen-tympani. Thrombus in lateral sinus. Pus under dura-mater at sinus.

*Case 166.*—*Edinburgh Medical Journal*, November, 1879. Treated by Kirk Duncanson. Male, age thirty-seven. Left ear. Acute otorrhœa; swelling

BOSIS OF THE CEREBRAL VEINS AND SINUSES FOLLOWING EAR DISEASE.

| Present History. | Result. | Autopsy. |
|---|---|---|
| Pain over mastoid; temp. 103°; pulse 92°; drum-head injected; perforation of drum-head; violent pain in ear and head; painful mastoid; mastoid opened, pus liberated; transverse sinus seen at bottom of incision; later, violent pain in temporal region; divergent strabismus of l. eye; later, temp. 99°; pulse 55°; ptosis of l. upper lid; loss of sensibility over lower extremities; skull trephined at tempero-parietal region; dura tense; dura incised; puncture of temporal lobe; pus evacuated; improvement; later, became worse, coma, death. | Death | Pus at chiasm. Pus in lateral ventricles. Abscess in temporal lobe. |
| Pain, aural suppuration, vomiting; mastoid tender; temp. 101°; occasional unconsciousness; mastoid opened, pus evacuated; improvement; suspended breathing; artificial respiration; successful; mastoid opening enlarged; dura exposed; exploratory needle introduced, upward and backward; pus evacuated | Death | Abscess of cerebellum. Pus in sulcus for transverse sinus. Thrombus in transverse sinus. Necrotic opening through tegmen-tympani. |

and redness over mastoid; pus came from an opening one-quarter of an inch in front of the membrana tympanum; pressure over mastoid caused the pus to flow more freely. Operation refused. Death.

*Autopsy.*—Pus in arachnoid space all over the brain. Dura healthy. Upper anterior surface of petrous was carious.

*Case 167.—Archives of Otology*, April, 1892. Treated by C. Truckenbrod, of Hamburg. Male, age fifty-four. Left ear. Previous history, deafness and tinnitus for ten days.

*Diagnosis.*—Exudation in tympanum; made a paracentesis; removed serum. Later, a furuncle developed in the meatus; tympanic discharge became purulent. Later, caught cold; pain in ear and head; temp. 104°; enlarged opening in drum-head. Later, opening again enlarged; continued pain in head; vertigo; paresis of right facial; aphasia; temp. medium; paresis of right arm.

*Operation.*—Mastoid opened; no pus; no necrosis; roof of mastoid cavity opened; dura exposed; no pus; dura opened, and brain exposed; no pus; exploratory puncture made, forward and upward, and pus evacuated; orifice enlarged by knife, and drainage tube inserted; cavity antiseptically irrigated; iodoform gauze used. Recovery.

*Case 168.—Archives of Otology*, April, 1892. Treated by C. Truckenbrod, of Hamburg. Male, age twenty-nine. Left ear. Previous history not given. Pain over mastoid; temp. 103°; pulse 92°; drum-head injected; perforation of drum-head; violent pain in ear and head; painful mastoid; mastoid opened; pus liberated; transverse sinus seen at bottom of incision. Later, violent pain in temporal region; divergent strabismus of left eye. Later, temp. 99°; pulse 55°; ptosis of left upper lid; loss of sensibility over lower extremities. Skull trephined at tempero-parietal region; dura tense; dura incised; puncture of temporal lobe; pus evacuated. Improvement. Later, became worse; coma. Death.

*Autopsy.*—Pus at chiasm. Pus in lateral ventricles. Abscess in temporal lobe.

*Case 169.—Archives of Otology*, April, 1892. Treated by C. Truckenbrod, of Hamburg. Male, age twenty-five. Left ear. Previous history not given. Pain; aural suppuration; vomiting; mastoid tender; temp. 101°; occasional unconsciousness; mastoid opened; pus evacuated. Improvement. Suspended breathing; artificial respiration; successful. Mastoid opening enlarged; dura exposed; exploratory needle introduced, upward and backward; pus evacuated. Death.

*Autopsy.*—Abscess in cerebellum. Pus in sulcus for transverse sinus. Thrombus in transverse sinus. Necrotic opening through tegmen-tympani.

*(To be continued.)*

## BOOK REVIEWS.

TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION, Volume Tenth. Edited by J. EWING MEARS, M.D., Recorder of the Association. Philadelphia: 1892.

This volume of most excellent reports of papers read at the meeting of the Association, May 31, June 1, and 2, 1892, is singularly defective in making no mention so far as we can discover of the place where the meeting was held.

This volume is particularly valuable in that it contains a complete index of the papers read before the Association from its first organization to the last meeting.

Dr. P. S. Conner of Cincinnati, was the presiding officer at the late meeting.

Dr. Nicholas Senn, was elected President for the ensuing year, and Dr. J. R. Weist Secretary, but where and at what date the book before us does not tell.

A MANUAL OF MEDICAL JDRISPRUDENCE. By ALFRED SWAIN TAYLOR, M.D., F. R.S., revised and edited by THOMAS STEVENSON, M.D., London. Eleventh American edition with citations and additions from the twelfth English edition, by CLARK BELL, Esq., President of the American International Medico-Legal Congress of 1893. 787 pages, 56 illustrations. Price, cloth, $4.50; sheep, $5.50. Lea Brothers & Co., Phila., 1892.

We have examined the above entitled work with more than usual interest, not only on account of the fact that it has gone through numerous editions and is well-known to the profession; but because in the preface of the learned American editor it is stated that much new matter has been added and a large number of authorities cited to aid council in preparing briefs, etc. A careful examination of the work shows that its value has indeed been enhanced, but we confess to a feeling of disappointment that the work has not been more thoroughly and exhaustively done. The author and English editor have cited very few American authors and those not always correctly. While perhaps this was to be expected of English authors, still the profession have a right to expect that an American who holds himself out as a member of so many learned societies as does Mr. Bell on the title-page, should remedy this defect and relieve the book of its insular character. The legal side of the editorial work has been more thoroughly done than the medical side, which is what one would naturally expect considering the fact that the editor is not a medical man. We are constrained to state, however, that much matter is included in the legal notes which, while valuable and interesting has no earthly bearing upon the subject of medical jurisprudence. This is notably the case with the note on expert evidence beginning on page 52, in which we learn among many other interesting but irrelevant things, that "masters of vessels or experienced seamen may give their opinions on questions pertaining to nautical science or affairs," to sustain which proposition, which no rational man would ever think of disputing at the present day, no less than twenty-two cases are cited.

On the other hand matters which, by reason of their importance merit an exhaustive treatment are merely touched upon or not even mentioned. The subject of malpractice for example, receives at the hands of the American editor only one and one-half pages, and even these show traces of hasty preparation.

References to American medical literature which abound in valuable and instructive cases (not in courts of law) are very few, so few as to lead one to believe that the field of periodical literature has not been explored by the editor.

For example, the subject of hypnotism is not mentioned in the index, and we have not found it treated in the text. Instances like the above might be multiplid.

Nothwithstanding all these indications of hasty or inadequate preparation of this edition, the work is still valuable but by no means so valuable as the profession have a right to expect.

A MANUAL OF ORGANIC MATERIA MEDICA, being a guide to materia medica of the vegetable and animal kingdoms, by JOHN M. MAISCH, Ph. M., Phar., D., fifth edition with two hundred and seventy illustrations. Philadelphia; Lea Brothers & Co. 1892.

The fact that a fifth edition of this excellent manual has been called for within two years of the publication of the fourth, is sufficient to confirm the value value of the work. Indeed, it has been so frequently reviewed and has become such a standard text-book, that any extended comment at this time is needless.

The present edition differs mainly from the former in the incorporation of the added knowledge upon this subject in the last two years, and the use of accent marks upon the systematic names of plants and animals. Several illustrations have been added, partly replacing others, further eludicating structural peculiarities.

We cannot let the occasion pass without commending the superior quality of press work shown in this volume. The wood-cuts show with surprising clearness.

MATERIA MEDICA AND THERAPEUTICS. By L. F. WARNER, M.D. Philadelphia. Lea Brothers & Company.

This little work of 223 pages forms part of "The Students' Quiz Series," now being issued by Lea's. While we have heretofore expressed doubts of the value of works of this character they have undoubtedly come to stay. The work before us is admirably arranged in the form of questions and answers especially adapting it for the use of students classes in which one of their own number act as leader. It also facilitates brevity and conciseness of expression, a *sine qua non* in a work of this sort.

The author says that he has aimed at "a convenient and concise statement of the most important facts of materia-medica and therapeutics." A careful reading of his pages shows that he has achieved success upon these lines, and within the limitations imposed by the size of the work. There are important omissions, and perhaps we would differ from him in some of his conclusions but to state them would we think, be hypercritical in the light of our conflicting authorities.

WISCONSIN STATE MEDICAL SOCIETY, 1892.

The Annual Volume of Transactions of this organization indicates an excellent working Society. The papers show a spirit of research on the part of many of the members that is exceedingly commendable.

The President of the meeting was Dr. G. F. Witter, of Grand Rapids, and Dr. C. S. Sheldon, of Madison, the Secretary. For the ensuing year Dr. B. T. Phillips, of Menominee, Mich., was elected President, and Dr. C. S. Sheldon, of Madison, re-elected Secretary. Number of active members 332.

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A PLEA FOR PUBLIC HEALTH IN VILLAGES.—When a fire breaks out in a village, every person considers it a duty to give a general alarm, and, especially, prompt notice of it to the fire departments, and all citizens coöperate for the speedy extinction of the fire. If this were not done, the property in the village would be quite generally endangered by the possible spread of the fire. Why is it that when a dangerous communicable disease breaks out—one which may spread and endanger quite generally the *lives* of persons in the village—there is, so frequently, no such general alarm and prompt notice to a well-organized department of the village government, and no general coöperation for the extinction of the disease? Is not a person's life of more consequence than his property? Is not the saving of the *lives* of their children of as much consequence to the inhabitants of the village, as the saving of their *property?* If each person were forced to answer this question, relative to his own children, I believe that he would feel like a degraded, miserly wretch if he did not promptly sacrifice his property in defense of the life of his child. Yet, collectively, the citizens generally do *not* do for the protection of *life*, what they do for the protection of *property*—they do not maintain a well-organized health department so generally as they do a well-organized fire department. I believe it is because they do not so generally know that lives may be saved by well-organized health departments, or because they do not know the value of human life to the community. —H. B. BAKER, M.D., in *Annals of Hygiene*.

THE PAN-AMERICAN MEDICAL CONGRESS.—The appearance of the preliminary announcement of the first Pan-American Medical Congress, to be held in Washington next September, is at once a promise, a guarantee, and a call to action. Few have an adequate conception of the magnitude of this undertaking, and of its significance to American medicine. It is very evident that it has now entirely passed the stage of doubt or indecision. The first thought that strikes one in glancing through the pamphlet of the preliminary announcement is as to the tremendous business and executive demands made upon the officers, and that so far have been splendidly met by the energetic Secretary-General, the President, and others. The officering of the long lists of the Sections has been a work of the greatest difficulty and finest judgment, and one can only marvel at the excellence of the results.

But it is now time for all American physicians to respond to the invitation and the opportunity in a spirit of patriotism to our country and loyalty to our profession. It is not now a question of the success of the Congress, but as to whether we shall or shall not give that early and effective sympathy that constitutes each of us a helper and a part of the success. The heavy expenses of organization, amounting to something like $5,000, must be paid by the advance registration-fees. The Secretary-General is Dr. Charles A. L. Reed, Cincinnati, Ohio, who, upon request, will forward copies of the preliminary announcement. The Treasurer is Dr. A. M. Owen. Evansville, Ind., to whom should be remitted the registration-fee.—*Medical News.*

IPECAC IN UTERINE INERTIA.—Drapes (*Der Frauenarzt*, March, 1892) says this remedy, in simple atony of the uterus, is a powerful agent in producing uterine contraction during the first and second stages of labor.

In general, two or three doses of from 10 to 15 drops of the wine of ipecac, given at intervals of ten minutes, produce, in a short time, marked activity of uterine action and a rapid birth. It is much better than ergot, as it does not produce tetanic contraction, but only induces normal and regular expulsive efforts.—*The Therapeutic Gazette*, July 15, 1892, p. 481.

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SATURDAY, NOVEMBER 26, 1892.

## THE ANTITOXIC PROPERTIES OF THE BLOOD OF CHOLERA CONVALESCENTS.

The researches of KLEMPERER (THE JOURNAL Oct. 29,) have shown, that while the blood serum of normal men possesses either no influence or but a very feeble one upon the toxicity of cultures of cholera bacilli a decided antitoxic effect is exerted by the blood of people who have been inoculated with cholera bacilli subcutaneously. The antitoxic effect of the blood serum of patients recovered from genuine cholera has now been tested by LAZARUS of Berlin, *Berliner klin. Wochenschrift*, Nos. 43 and 44, with remarkable results.

Our conception of the term immunity has gained in precision very much since clinical observations have been supplemented by laboratory work. From the earlier researches of PASTEUR on anthrax and chicken cholera it became evident that there may be different degrees of immunity. After BEHRING had discovered that an important—if not the most important factor in acquired immunity is the acquired antitoxic property of the blood, EHRLICH taught us how to measure this property and thus express the degree of immunity in terms of mathematical accuracy.

KLEMPERER found it possible to influence the economy by his inoculation with cholera bacilli under the skin to such an extent, that one-half to one cc. of the person's serum injected into a guinea pig protected the latter against a fatal dose of a cholera culture. In the natural immunity following convalescence from an attack of cholera LAZARUS found the antitoxic effect of the serum 5 to 10,000 times as great as in the blood of the vaccinated person. One-tenth of a milligram of the serum was sufficient to protect a guinea-pig against death while ½ m. gr. prevented even any transient effect of an otherwise fatal dose of virulent cholera-culture.

This marvellous antidotal influence occurs only if the protective injection is made before the poisonous cholera-toxines are injected into the peritoneum. After the introduction of the cholera poison protection can be had only from enormous doses of serum up to 1 cc. and from these only if they are given during the incubation period of the first few hours. After the effects of the cholera poison had become manifest by the lowering of the animal's temperature, no curative results could be obtained from serum in any quantity.

The author suggests that a trial might be made in man to utilize the antitoxic properties of the blood of convalescents during the premonitory diarrhœa of cholera, and before systemic poisoning has set in.

## THE SURGICAL TREATMENT OF URETERAL WOUNDS.

There are but few reported cases of uncomplicated wounds of the ureters, though one or both of these ducts have not unfrequently been injured in abdominal operations on the ovaries, uterus, or vagina, GUSSEROW and other authorities stating that this accident has happened to most surgeons that have frequently performed vaginal hysterectomy; and traumatism during parturition has been the most frequent cause of uretero-vaginal or uretero-uterine fistula.

DR. F. KAMMERER published in the *New York Medical Journal* for July 2, a contribution to the treatment of ureteral fistula, in which he reported two cases of uretero-abdominal fistula caused by difficult abdominal operations, in each of which he subsequently successfully performed nephrectomy. The latter operation, first performed by SIMON for the cure of ureteral fistula, has been performed about an equal number of times for the cure of uretero-abdominal and uretero-vaginal or uretero-uterine fistula. Of the thirteen recorded cases of nephrectomy for ureteral fistula collected by KAMMERER, only two resulted fatally; one death being due to insufficiency of the remaining kidney.

When the ureter has been divided by a stab or gunshot wound, or accidentally in an operation, what should the surgeon do? In the recently published *American Text-book of Surgery*, the authors recommend that the divided or torn end should be brought to the surface of the loin or of the vagina and sutured there. This would probably entail a subsequent nephrectomy, an operation that these authorities countenance by referring to it as the measure for cure of the fistula, although they do not recommend its immediate performance so as to prevent the formation of a fistula.

Transplantation of the end of the ureter to the abdominal wall is not a simple operation, and KAMMERER proposed sometime ago that the renal end of the ureter should be ligated or secured by forceps. He has since withdrawn his recommendation of a liga-

ture, because GUYON'S experiments have demonstrated that, even under ordinary conditions, it will produce the symptoms of renal retention; the ligature causing by its firm constriction of the ureter the same reflexes that an impacted ureteral calculus occasionally excites. Closure of the renal end of the ureter by forceps, with an iodoform gauze tampon thereabout, would permit the removal of the forceps in the event of renal symptoms developing; and if the tampon had not excited adhesions that completely shut off the peritoneal cavity, any leakage of urine would tend to the surface by means of the tampon.

Instead of transplanting the ureters to the loin or vagina, DR. R. HARVEY REED has suggested in the *Annals of Surgery* for September, that in injuries of the ureters in consequence of ovarian or other neoplastic adhesions, or in case of disease of the bladder, the ureters should be implanted in the rectum. He has made some interesting experiments on animals to determine the feasibility of this procedure, and his experiments show that the unilateral implantation of the ureter into the rectum was a possible and practical surgical procedure, though bilateral implantation always terminated fatally in the animals experimented on. He found that the rectum readily accommodated itself to the presence of urine, and that the stools were not necessarily liquid or frequent. He believed that the experiments showed that it was a safer plan to implant a ureter than to perform nephrectomy. This operation has yet to be performed on the human subject, though DR. REED'S results commend it in preference to lumbar or vaginal implantation of the ureters, and it possesses the additional desirability of dispensing with a subsequent nephrectomy.

While nephrectomy has been immediately and successfully performed in such cases of ureteral injury, still such an additional operation is not without risk when a patient has just been subjected to the shock of a capital abdominal operation. And as DR. KAMMERER very pertinently suggests, while the increased demands upon the remaining kidney during the days following nephrectomy may be easily responded to when no other complication is present, they might prove too great when the patient is already struggling against the shock consequent upon a severe and protracted laparotomy. Furthermore, the operation of nephrectomy is far from being one of the most favorable procedures in surgery; though the condition of both kidneys may be ascertained to an extent at the laparotomy during which the ureter is injured.

The improbability of securing immediate union in a severed ureter in which there is no loss of ureteral tissue, would make the risk of intra-peritoneal extravasation of urine too great to attempt to suture the severed ends of that duct.

For the reasons above mentioned it may be concluded that in wounds of the ureter complicating abdominal operations immediate nephrectomy is undesirable, although its performance is not unjustifiable in traumatic injuries of the ureter *per se*.

That while the lumbar or vaginal implantation of the ureter has been successfully performed, secondary nephrectomy has often been resorted to for the cure of the troublesome ureteral fistula. And that the successful implantation of the severed ureter into the rectum, offers a measure that avoids the inconveniences and dangers of each of the foregoing procedures.

## "PYOSALPINX."

"Pelvic cellulitis" existed theoretically, based upon purely clinical phenomena, and the term has almost disappeared from the modern nomenclature of disease, while the condition so long described as such has at last received its proper place in surgical science.

That pelvic cellulitis may occur cannot be denied, but that it is rare, even as a result of post-puerperal infection, has been proved by hundreds of accurate observations, both at the operating table and by post-mortem examinations. The classical essays of BERNUTZ and GOUPIL lay long forgotten under the rubbish of dogmatic and incorrect teaching, until the master mind of LAWSON TAIT, aided in Germany by HEGAR and MARTIN, and in America by JOSEPH PRICE, forced the attention of the profession to the exact pathology upon which "pyosalpinx" is founded. It is fortunate for the peace of mind of the common sense practitioner that his imagination is no longer taxed with a mysterious cellular absorption, for he can now readily understand an infection of a septic, gonorrhœal or tubercular nature, traveling by continuity of tissue from the endometrium to the tube, and too frequently he has had reason to regret a careless interference with the aseptic uterine cavity.

Nature in her effort at self-protection employs here, as with foreign bodies generally, a combination of the three methods, absorption, encapsulation and extrusion, and while she may be more or less successful in her effort, too frequently the result is disastrous to the life or health of the patient. Absorption and resolution of a mild infection of the tube without the formation of a pyosalpinx may take place, but often destroys the ciliated epithelium, thus rendering possible a future ectopic pregnancy. An encapsulation of the pus in the tube forms a pyosalpinx which is a source of constant danger through recurring attacks of pelvic peritonitis, or a possible perforation into the free peritoneal cavity. If unilateral, and pregnancy occur, the abscess may be ruptured during parturition, causing a fatal puerperal fever.

Extrusion of this pus by means of adhesions to the bowels and intestinal opening, either spontaneous or by the surgeon's knife, is usually an unfortunate occurrence through the resulting fecal contamination and inefficient drainage, while vaginal or more superficial openings may leave a suppurating stump of an ovary and tube which, like a sequestrum of necrosis in an involucrum of healthy bone, keeps up a chronic discharge and undermines the health of the victim. These undeniable facts lead to but one conclusion, that the early and complete removal of a pus tube by cœliotomy is surely indicated. The mortality of JOSEPH PRICE of less than 3 per cent. in his magnificent work is, however, almost unique, and the average death-rate, as given by MCLAREN, of St. Paul, in a recent paper, is about 9 per cent. in the hands of experienced operators. The plea for conservative treatment then, from the standpoint of immediate mortality of the operation itself, rather than the sentimental one so frequently advanced as to the removal of a useless and dangerous organ, is worthy of the utmost attention. The claim of electricity depends upon a basis of inaccurate diagnosis. Vaginal puncture is a blind and uncertain procedure, and as a pus tube frequently contains two or three complete strictures separating it into several pockets, the chance of emptying them all is small at best. The new treatment of curetting and iodoform gauze pack of the uterus, so useful in an endometritis, is an exceedingly insecure method of treatment in pyosalpinx. A very small number of well authenticated cases in which a pyosalpinx or cystic tube has been emptied into the uterus, cannot counterbalance the danger of such operative interference. The large majority of reports in which there has been a supposed uterine drainage of a pus tube, have been shown by J. BLAND SUTTON to be merely the discharge of retained uterine secretions, and in any event, the benefit of such practice is probably not greater than follows free purgation. Any operation which opens the abdomen and then does not remove the pyosalpinx does not deserve the name of conservative, and is not good surgery, whether done by stitching the tube to the incision, or the late recommendation of the French to make a vaginal hysterectomy and leave the pus tubes to drain.

### RAILWAY SURGERY AT THE PAN-AMERICAN MEDICAL CONGRESS.

A Section of Railway Surgery of the Pan-American Medical Congress has been organized with DR. C. W. P. BROCK, of Richmond, Virginia, as Executive President. A full list of officers has been provided for each of the constituent countries. At the Eleventh Annual Meeting of the Wabash Railway Surgical Association—the first organization of the kind—DR. C. B. STEMEN, of Fort Wayne, was by unanimous resolution, requested to prepare a paper on "Organized Railway Surgery," and read the same before the Section on Railway Surgery of the Pan-American Medical Congress. At the same meeting DR. HAL C. WYMAN, of Detroit, offered the following, which was unanimously adopted:

*Resolved*, That each member of this Association solicit his Congressman to interest himself in legislation in favor of the Pan-American Medical Congress.

THE SAMUEL D. GROSS PRIZE, PHILADELPHIA ACADEMY OF SURGERY.—The first quinquennial prize of one thousand dollars, under the will of the late Samuel D. Gross, M.D., will be awarded in 1893. The conditions annexed by the testator are that the prize "shall be awarded every five years to the writer of the best original essay, not exceeding one hundred and fifty printed pages, octavo, in length, illustrative of some subject in Surgical Pathology or Surgical Practice, founded upon original investigations, the candidates for the prize to be American citizens." It is expressly stipulated that the successful competitor shall publish his essay in book form, and that he shall deposit one copy of the work in the Samuel D. Gross Library of the Philadelphia Academy of Surgery. The essays, which must be written in the English language, should be sent to Dr. J. Ewing Mears, 1429 Walnut St., Philadelphia, before June 1, 1893. Each essay must be distinguished by a motto, and accompanied by a sealed envelope bearing the same motto, and containing the name and address of the writer. No envelope will be opened except that which contains the successful essay. The committee will return the unsuccessful essays if reclaimed by their respective writers, or their agents, within one year. The committee reserves the right to make no award if the essays submitted are not considered worthy of the prize.

## SELECTIONS.

BIOLOGIC THERAPEUTICS.—Medicine has entered upon a new era in therapeutics, which may appropriately be designated biologic. The scientific world greeted with cordial admiration the synthetic products of the chemist's skill, but we stand to-day on the threshold of developments the magnitude of which is beyond comprehension. Since the birth of the young science of bacteriology practical minds have assumed an expectant attitude. Skepticism has largely given way to confidence, and excessive enthusiasm to a calmer judgment. It soon became apparent, in a manner and to a degree in which it had never been before, that many diseases carried with them the elements of their own cure. Of course, it had been recognized that certain diseases are self-limited, and the phenomena of natural and acquired immunity were duly appreciated; but it required the knowledge gained by the advances in bacteriology to afford a rational explanation of these various phenomena. There is yet much to learn. The beginning has but been made. Enough, however, has been seen to teach that disease has its chemistry, and that the treatment of the future will depend upon a knowledge of this fact and the application of chemic laws. It is not too much to hope that the treatment of the future will largely be specific, that is, in the employment of a definite remedy in the treatment of a given affection, just as mercury and iodine are to-day employed in the treatment of syphilis, quinine in the treat-

ment of malarial disease, and salicylic acid in the treatment of rheumatism. The groundwork of this new system of therapy has been already laid by Pasteur as to hydrophobia, by Koch in regard to tuberculosis, by Behring, Kitasato, Wasserman, and Ehrlich as to diphtheria, by Tizzoni, Centanni, and Cattani as to tetanus, by the Klemperers as to pneumonia, by Ferran, Haffkine, and Klemperer as to cholera.

Stern (*Drutsche medicin. Wochenschr.*, 1892, No. 37, p. 827) has made an interesting contribution to this subject. He had previously demonstrated that human blood-serum possesses the property of destroying the bacilli of typhoid fever, He now endeavored to determine if the bactericidal activity of the blood-serum to the bacillus of typhoid fever is increased in persons that had recovered from an attack of that disease; if the blood of such persons had any curative action upon animals inoculated with the bacilli of typhoid fever; and if this blood has the property of neutralizing the poisons generated by the bacilli of typhoid fever. Seven cases were examined, six at intervals of from five days to five and a half weeks after defervescence had set in, and one, who was under treatment for sciatica, seventeen and a half years after an attack of typhoid fever. It was found that the bactericidal activity of the blood-serum to the bacilli of typhoid fever was distinctly diminished in persons recently convalescent from typhoid fever, in comparison with the conditions present in persons that had never had typhoid fever. It was further found that, while the serum of a healthy person exerted no modifying influence upon the toxicity of boullion-cultures of the bacilli of typhoid fever with which it was mixed, the serum of persons recently convalescent from typhoid fever exerted a distinctly modifying effect of an attenuating character. It was also demonstrated that a filtered extract of sterilized cultures of the bacilli of typhoid fever could be safely injected in doses otherwise lethal, if previously admixed with the blood-serum of persons recently convalescent from typhoid fever.

From the foregoing it is clear that the protective influence of the blood-serum of persons recently convalescent from typhoid fever is not dependent upon a destructive action upon the bacilli themselves. It will not do, however, at once to jump to the conclusion that the serum neutralizes the toxic products of the bacilli. It is also possible that the serum renders the animal organism less susceptible to the influence of the toxic products. This doubt would be removed if it were shown that the diminution in the toxicity of a mixture of the serum and the sterilized extract were progressive.

The observations here recorded are most interesting, and are deserving of thoughtful consideration. They should be repeated and extended. They seem to bring us nearer to a correct conception of natural immunity and natural cure. —*Medical News.*

WHY WE SHOULD HAVE A MEDICAL OFFICER IN THE PRESIDENT'S CABINET.—During the recent assault made upon the Atlantic fortifications of the great United States, the country was in danger of utter destruction, and every individual in peril of his life on account of the clash of authority which occurred between the federal and city authorities. Fortunately the federal officers gained the day, and great credit is due to them as well as to the Health Officer of New York City in keeping out the merciless foe of mankind. Although cholera entered New York, it can scarcely be said to have gained a foothold, and yet we shall await anxiously the developments of the coming spring, as it is now conclusively shown that cholera germs may lie dormant for months, and reappear under favorable climatic and filth influences.

But why have a repetition of so unseemly a disagreement? Why endanger the lives of sixty-five millions of people by a clash of any sort? Why not establish a medical cabinet officer as other civilized nations have, who shall have supreme authority in all such matters? We can see no more valid objection to a Minister of Public Health than we do to a Secretary of State or a Secretary of the Navy, for are not our commercial interests the lives of millions, as well as our very existence threatened on such critical occasions as we recently witnessed in New York harbor? Why, therefore, should we not urge our claims of protection from a foe more deadly than the invading Britons. By all means, let us have a medical officer of public health in the cabinet. Let him be well provided with a small army of medical men to guard us against further invasions. The health of our nation should be of supreme importance. It is estimated that the monetary loss to New York City alone by the present visitation of cholera was over 2,000,000 dollars! This fact alone should be sufficient to call the attention of our law-makers in Washington to the importance of the subject. Let us hope the matter will receive the attention of our next Congress. Surely no measure of greater importance can be introduced.—*Pacific Medical Journal.*

## MISCELLANY.

WANTED.—Will pay 10 cents per copy for the following numbers of THE JOURNAL: Vol. 2, No. 4, Jan. 26; No. 19, May 2, 1884. Vol. 5, No. 2, July 11; No. 3, July 18, 1885. Vol. 6, No. 6, Feb. 6, 1886. Vol. 3, No. 4, July 26, 1884. Vol. 4, No. 22, May 30, 1885; No. 23, June 7, 1885; No. 24, June 14, 1885; No. 25, June 21, 1885; No. 26, June 28, 1885. Also, Vol. 12, No. 3, Jan. 12, 1889.. Vol. 12, No. 3, Jan. 19, 1891.

OFFICIAL LIST OF CHANGES in the Stations and Duties of Officers Serving in the Medical Department, U. S. Army, from November 12, 1892, to November 18, 1892.

First Lieut. Henry R. Stiles, Asst. Surgeon (recently appointed), is assigned to duty at Jefferson Bks., Mo.

First Lieut. Paul F. Straub, Asst. Surgeon (recently appointed), is assigned to duty at Ft. Riley, Kan.

First Lieut. Francis A. Winter, Asst. Surgeon, is relieved from duty at Ft. Riley, Kan., and is assigned to duty at Ft. Wingate, N. M.

First Lieut. A. E. Bradley, Asst. Surgeon, is relieved from duty as attending surgeon, Hdqrs. Dept. of the Platte, Omaha, Neb., and is assigned to duty at Ft. Sully, S. Dak.

Capt. Benjamin Munday, Asst. Surgeon, is relieved from duty at Ft. Sully, S. Dak., and is assigned to duty at Ft. Niobrara, Neb.

First Lieut. Harry M. Hallock, Asst. Surgeon (recently appointed), is assigned to Ft. McPherson, Ga., for duty at that post.

First Lieut. Robert S. Woodson, Asst. Surgeon, is relieved from duty at Ft. McPherson, Ga., and is assigned to duty at Ft. Barrancas, Fla.

First Lieut. George J. Newgarden, Asst. Surgeon (recently appointed), is assigned to duty at Ft. Sheridan, Ill.

First Lieut. Charles F. Kieffer, Asst. Surgeon, is relieved from duty at Ft. Sheridan, Ill., and assigned to duty at Ft. Meade, S. Dak.

OFFICIAL LIST OF CHANGES in the Medical Corps of the U. S. Navy, for the Week Ending November 19, 1892.

Asst. Surgeon Geo. T. Smith, from Coast Survey Str. "Hassler," and to the "Vermont."

P. A. Surgeon C. H. T. Lowndes, from the U. S. receiving ship "Vermont," and to the "Hassler."

P. A. Surgeon W. F. Arnold, to hold himself in readiness for orders to the U. S. S. "Monterey."

P. A. Surgeon H. N. T. Harris, to hold himself in readiness for orders to the U. S. S. "Bancroft."

Surgeon G. F. Winslow, to hold himself in readiness for orders to the U. S. S. "Monterey."